PERINATAL CALF MORTALITY IN THE KANSAS FLINT HILLS

by

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INTRODUCTION

Production efficiency is the most important factor in determining profitability of a commercial beef cow-calf enterprise. The goal is to obtain a live marketable calf from every cow bred each year. A major limitation in realizing this goal is perinatal calf mortality (PCM). Cow-calf production efficiency is also limited by infertility. When the PCM and infertility incidence in a herd is compared to the total number of heifers and cows bred, a useful assessment of the herd's production performance can be made. The percent loss in production efficiency results in a waste of capital, resources, time, and labor.

The incidence of infertility in beef herds has been established as effective reproductive management programs have been increasingly employed during the past 15 to 20 years by using rectal palpation. These programs have effectively reduced and prevented the incidence of infertility from increasing.

Studies and surveys over the past 60 years reveal that perinatal mortality figures have not significantly improved inspite of the intensity of beef cattle production being increased. In light of advances in technology, nutrition, husbandry, infectious disease control, preventive medicine, antibiotics, and therapeutic agents, this lack of significant improvement is embarrassing to say the least. Only in

the last decade have we earnestly started to look towards preventive medicine as a way of improving PCM. The key to successful reduction of PCM is prevention, not treatment.

Preventive medicine alone is only moderately successful if the perinatal problems are not accurately identified and defined. There has been a significant reduction in perinatal mortality in man because the problems encountered by human neonates have been fairly well defined and appropriate corrective methods utilized. Recently in veterinary medicine, new insights into factors influencing calf mortality have been provided by systematic necropsy findings correlated with fetal, natal and neonatal problems and their time-of-death. It is unlikely that PCM losses will be reduced until the problem is clarified and all factors defined.

This study was conducted to define perinatal problems in selected beef herds in the Kansas Flint Hills. The objectives of the project were to:

1) Determine and clarify the causes of PCM;
2) Establish in individual contract herds the incidence and distribution of PCM based on time-of-death classification;
3) Establish the "normal" incidence and distribution of PCM in the area from the individual herds;
4) Determine the percent of PCM within the three major causes of death: infectious, non-infectious, and undetermined;

5) Clarify each major cause of death category by identifying the most common causes; and

6) Determine if diagnostic efficiency is affected by the number and types of tests performed, and which tests are the most diagnostic.

I. REVIEW OF LITERATURE

This review has been confined primarily to the incidence, causes, diagnosis and management of infertility and perinatal calf mortality of beef breed herds. Information concerning dairy herds is presented only in areas lacking information on beef calves. The reasons for this approach are based on the economic importance that beef cattle have on agriculture in the United States, particularily the Great Plains area, and the following differences between beef and dairy herds:

1. Beef cattle calving season is more limited (45 to 120 days during spring or fall) that that of dairy cattle;
2. Beef calves are influenced more by their dams as they remain with them longer than dairy calves;
3. Beef herd nutrition is primarily from grazing range land and low quality crop residues with little supplementation;
4. Beef herds are exposed to more environmental stresses and influences that play a major role in production efficiency;
5. Husbandry practiced by beef producers is more extensive and cost-oriented;
6. Beef cattle ranks as the largest segment of United States agriculture and is the most important industry in mid-west states as beef cattle are the primary source of income for the Great Plains livestock producers (N.C.A., 1982).

DEFINITIONS

To prevent confusion about the terms used, the recommendations by the Committee on Bovine Nomenclature will be followed (Hubbert et al., 1972).

Pregnancy Wastage: Reproductive failure regardless of cause, synonymous with reproductive dysgenesis -- primarily infertility and/or perinatal mortality.

Infertility: Temporary reproductive failure, primarily conception failure and/or early embryonic mortality.

Conception Failure: Failure of fertilization of ova from any cause.

Embryonic Mortality: Death or loss of the conceptus during the embryonic period (approximately the first 45 days).

Perinatal Mortality: Death of fetuses and newborn calves occurring after the embryonic period, during birth, or during the first 28 days of life.

Fetal Death: Death of a fetus prior to complete expulsion or extraction from the dam. Fetal deaths are subdivided into antepartum and intrapartum (parturient).

Stillbirth: A clinical term simply meaning found dead at first observation, usually a fullterm dead fetus. The term is not sufficiently accurate to indicate the time-of-death without necropsy. It has been recommended that fetal death be substituted for stillbirth.

Abortion: Expulsion before fullterm of a conceptus incapable of independent life--includes embryonic and fetal losses.

Premature Birth: Expulsion before fullterm of a fetus capable of independent life.

Dystocia: Difficult or abnormal parturition.

Neonatal Death: Death of newborn calves occurring within the first 28 days of life. Neonatal deaths are subdivided into hebdomadal (under 7 days) and posthebdomadal (8 to 28 days).

PREGNANCY WASTAGE

Beef cattle pregnancy wastage (PW) has been surveyed by many over the past 60 years (Baker and Queensberry, 1944; Ensminger et al., 1955; Grunsell, 1956; McCormick et al., 1956; Kincaid, 1957; Woodward and Clark, 1959; Rice et al., 1961; Wiltbank et al., 1961; Koger et al., 1967; Nat. Acd. Sci., 1968; Young, 1968; Rice and Wiltbank, 1972; Laster et al., 1973; Laster and Gregory, 1973; Young and Blair, 1974; Philipsson, 1976; Tayler, 1976; E.E.C., 1979; Patterson et al., 1979; Thornberry, 1979; Khera, 1981; Roche, 1981; Cope, 1982; Drost et al., 1982). Due to lack of standardized definitions for many terms used to evaluate pregnancy wastage, comparison between many studies is difficult. It is, however, easy to see that inspite of advances in nutrition, husbandry, infectious diseases, and therapeutic agents, the incidence of perinatal calf mortality has not appreciably decreased (Roy, 1980).

Total beef cattle pregnancy wastage is composed primarily of reproductive dysgenesis resulting from infertility and perinatal calf mortality. Infertility and perinatal calf mortality constitute the major losses in beef cattle (Nat. Acd. Sci., 1968; Oxender and Adams, 1979; Thornberry, 1979; Roche, 1981; N.C.A., 1982; Koch and Algeo, 1983). The estimated incidence of pregnancy wastage ranges from 5.7 to 41.5% of all cows bred with an average of 24%. Multiparous cows experience a lower incidence of PW than primiparous heifers (Young, 1968; Laster et al., 1973; Laster and Gregory, 1973; Patterson et al., 1979). Other factors influencing PW are breed, type of operation, geographic area, infections, nutrition, behavior, toxic agents, dystocia, and season (Dennis, 1979, 1980a,b,c, 1981).

PW is influenced by events occurring during four periods: embryonic, fetal, natal, and neonatal. In order to evaluate PW in a herd, it is necessary to understand and to be able to detect the incidence and causes of reproductive losses within the major areas comprising the total PW.

1. Infertility

Infertility is temporary reproductive failure (Hubbert et al., 1972). It can result in delayed calving and long breeding seasons, or more commonly open females (not pregnant) as in herds with structured breeding programs. The reported range of infertility in beef herds is 2.3 to

22.0% of all females bred, average incidence of approximately 16.8% (Ensminger et al., 1955; Nat. Acd. Sci., 1968; Young, 1968; Young and Blair, 1974; Patterson et al., 1979; Cope, 1982).

Infertility is usually detected clinically at pregnancy diagnosis. It results from either conception failure and/or early embryonic mortality (Hawk, 1979). To differentiate between the two is difficult without an adequate history and physical examination. Causes of conception failure and embryonic mortality are many and include infectious, non-infectious, genetic and undetermined.

2. Perinatal Calf Mortality

Perinatal calf mortality (PCM) makes up the remainder of the losses in the total PW of a herd. Perinatal losses extend from the beginning of the fetal period, approximately 42 to 45 days of gestation, through parturition, and for the first 28 days of life (perinatal period). The reported incidence of PCM in beef herds is 3.5 to 25% of all females diagnosed as being pregnant, with an average of approximately 8.6% (Nat. Acad. Sci., 1968; Young, 1968; Laster and Gregory, 1973; Young and Blair, 1974: Oxender and Adams, 1979; Patterson, 1979; Thornberry, 1979; Dennis, 1980a; Khera, 1981). PCM is more costly to producers than infertility because of the increased time and resources that are

wasted during development of the fetus or calf before it is lost. Factors effecting PCM and the sequellae are summarized in Fig 1.

All perinatal losses should be assessed on a herd basis (Dennis, 1979). Only during the last 20 years have ways been found for standardizing PCM by systematic necropsy so that herds in any area can be compared accurately (Young and Blair, 1974). Perinatal deaths can be effectively divided into: fetal or antepartum, natal or partum, and neonatal or postpartum.

McFarlane (1965) devised a practical system based on gross signs for classifying time-of-death (TOD) related to birth in lambs. He divided perinatal deaths into 21 classes: antepartum 2, partum 7, and postpartum 12 (Table 1). Some researchers have adopted this systematic approach for investigating calf mortality (Young and Blair, 1974; Dennis, 1980a; Kavasnicka, 1984). Criteria for classifying calves into one of the three main categories are:

Fetal Deaths--Characterized by absence of any signs of viability during or after parturition or abortion and gross signs of death such as edema (clear to blood-tinged), autolysis, or mummification.

Natal Deaths--Fetal death during parturition characterized by positive signs of viability (e.g. functional heart producing localized edema, and possibly some lung aeration) and absence of signs of survivability (e.g. renal cortical autolysis). Relationship between

duration of parturition and TOD is based on the degree of local edema indicating the length of time the fetus survived, and renal cortical autolysis indicating how long the fetus has been dead.

Neonatal Deaths--Characterized by positive signs of having survived birth with a functional heart, indicated by a clot in the umbilical arteries. Neonatal deaths are subdivided into immediate, delayed or late based on presence or absence of signs of breathing, walking, ingestion and absorption of milk, and metabolism of body-fat.

EMBRYONIC PERIOD

Approximately 27% of all first breedings of healthy cows and fertile bulls are lost (David et al., 1971). Roche et al. (1981) identified that fertilization failure can account for up to 20% of infertility in a herd.

1. Non-infectious Infertility

Non-infectious and undetermined factors appear to be the major causes of fertilization failure. Nutrition is probably the most important environmental influence affecting reproduction (Cope, 1982). Other known causes include poor fertility and poor libido of bulls, failure of sperm transport or survival, age of dam, inbreeding, reproductive tract disorders, developmental defects, ovarian tumors and

cysts, hormonal disturbances, seasonal influences, heat stress, ingestion of plant estrogens, and nutrient deficiencies such as phosphorus, protein, vitamin A, cobalt, copper and iron (Roberts, 1971; Zemjanis, 1980; Miller, 1982; Francos and Mayer, 1983).

Embryonic mortality can occur anytime from conception to approximately 45 days of gestation with most occurring with hatching of the blastocyst and initiation of elongation and commencement of implantation (Roche, 1981). The physiological explanation for high losses at this time is not clear. A large portion of the losses are probably genetic and have been termed "the elimination of faulty genetic experiments at low biological costs" (Miller, 1982). Before day 14, the developing bovine zygote is resistant to teratogens but is susceptible to genetic mutations and chromosomal aberrations (Leipold and Dennis, 1980).

2. Infectious Infertility

Infectious causes of embryonic mortality have long been a favorite topic for research as many agents produce infection of the female genital tract (Roberts, 1971). Their primary effect is inhibiting attachment of the embryo, resulting in mortality. The major infectious diseases producing embryonic mortality in the United States are venereally transmitted and the list includes:

1) Campylobacteriosis
2) Trichomoniasis

3) Brucellosis

4) IBR-IPV

5) Granular venereal disease

6) Miscellaneous

1) Bovine Genital Campylobacteriosis (vibriosis): Campylobacteriosis is due to a Gram-negative bacterium, Campylobacter fetus ss venerealis. First described by M'Fadyean and Stockman in 1909, it has become the most important cause of infectious infertility in beef cattle in the western range states (Roberts, 1971; Hoerlein, 1980). Tentative diagnosis is usually made at pregnancy examination by rectal palpation that reveals great variation in the stage of pregnancy in the herd. The pregnancy rates in seasonally bred herds by natural service will vary from 30 to 85% (Hoerlein, 1980). A low incidence of mid-trimester abortions (< 10%) may be associated with campylobacteriosis.

Campylobacteria usually invade the uterus about 7 days after natural service by an infected bull and usually persist in the uterus for about 13 weeks until local immunity develops and infection is eliminated. Rarely does an infected cow carry infection through a normal gestation period (Roberts, 1971). The immunity is not strong enough to last much longer than several days or weeks (Hoerlein, 1980). Although bulls do not develop immunity, young bulls under 5 years of age are difficult to infect experimentally. They may carry campylobacteria from cow to cow during the breeding season and then rapidly become free of infection.

In bulls over 5 years of age, the disease may become chronic and campylobacteria may perisit within the prepuce for years. Campylobacteriosis can also be spread by artificial insemination with semen from infected bulls.

Diagnosis is made by demonstrating the organisms by phase contrast microscopy, stained smears, or immunofluoresence, and by confirming it by culturing cervical mucus. If mucus samples cannot be cultured within 6 hours after collection, the plastic collection pipettes should be frozen with dry ice immediately (Hoerlein, 1980). A good method for diagnosing infection in a suspect bull is to breed him to virgin heifers and culture the cervico-vaginal mucus.

Artificial insemination and vaccination of all cows and heifers with an oil adjuvant bacterin are the two most common and most effective means of controlling and preventing bovine campylobacteriosis (Hoerlein, 1980). Vaccination of bulls appears to be effective in modifying the carrier state but effective elimination can only be accomplished by treatment with dihydrostreptomycin.

2) Trichomoniasis: A protozoal disease due to Trichomonas foetus, causes infertility, early abortion, and pyometra. The protozoan agent was discovered in the United States first by Emmerson in 1932, and since then, it has been found in most states in the United States (Roberts, 1971). Similar to campylobacteriosis, the disease is usually diagnosed during pregnancy evaluation with similar fetal age variation. Cases of pyometra with a fluidy consistency somewhat like "potato soup" may be observed.

After infection by an infected bull, the protozoan localizes in the secretions of the vagina, uterus, and oviducts. In some but not all infected females, a muco-purulent vaginitis or endometritis or vagino-endometritis may occur. This is usually detectable approximately 50 days postservice (Parsonson et al., 1976). Once infected, cows remain infertile for 2 to 6 months, develop immunity, and usually conceive and carry to term (Abbitt, 1980). The duration of immunity is variable. The disease is asymptomatic in bulls with bulls older than 4 years of age tending to become permanent preputial carriers. younger bulls either spontaneously recover or do not contract the disease (Abbitt, 1980). Trichomoniasis also can be spread by artificial insemination with infected semen.

Isolation of T. foetus is necessary to confirm the diagnosis and this is best accomplished by microscopic examination of preputial and vaginal fluids (Abbitt, 1980). Control measures include sexual rest, culling infertile cows, artificial insemination, and the use of bulls less than 4 years old. Various systematic and local anti-protozoan agents are effective in treating individual cases but are too expensive to be used on a herd basis.

3) Brucellosis: Although classically known as a major cause of abortion, there is evidence that uterine infection with Brucella abortus plays a definite role in infertility (Roberts, 1971). Transmission via ingestion is the most

common means of spreading brucellosis but venereal transmission may play an important role in infertility. Experimentally venereal transmission of brucellosis from infected bulls to susceptible cows is difficult, however, artificial insemination with semen from brucella-infected bulls produces brucellosis in inseminated females. The resulting uterine infection induces infertility for 3 to 12 months (Roberts, 1971).

Miller and Graves (1932) reported that brucella-positive cows averaged 2.8 services per conception following normal calving and 3.6 services per conception following abortion. Since the beginning of the federal brucellosis eradication program, the incidence of brucellosis has markedly declined. However, due to the severe economic results it can have on a herd or area it should always be considered until it is eradicated from the country.

Herds with brucellosis experience all the manifestations of the disease. Serologic diagnosis is the most common method of detecting infected females. Even though sexual rest and intrauterine treatment help, it is recommended that infected cows be sold as they may be brucella-shedders for life.

4) IBR-IPV Complex: Infectious bovine rhinotracheitis-infectious pustular vulvovaginitis (IBR-IPV) is a reproductive disease caused by the IBR respiratory virus. The respiratory form of the disease, however, rarely accompanies the genital form (Roberts, 1971). The IBR virus is endemic

in the western range states and the genital form may be more prevalent than thought due to its insidious nature and the extensive husbandry practiced on most range land ranches.

Clinically, an infected female exhibits vulvar edema, pain on urination, and a serous to purulent vulvar discharge. On inspection of the vulva and posterior vagina, white circumscribed papules and pustules are observed that may coalesce to form membranes that leave mucosal ulcers upon detachment (Kendrick et al., 1958). Other lesions reported are necrotizing endometritis and cysts on corpora lutea (Miller and Van DerMarten, 1984). The virus also produces balanoposthitis in bulls (Kahrs, 1980). Infection may be spread by natural service, artificial insemination with contaminated semen, and by breeding shortly after injection with a modified live IBR virus vaccine.

The disease has a rapid onset (3 to 6 days) and involves 60 to 90% of a herd (Roberts, 1971). Infertility is caused by reluctance of bulls or cows to mate because of local irritation and pain, or by embryonic death from necrotizing endometritis. In one study using infected semen, the estrous cycles were shortened to 11 to 15 days and only 1 of 12 heifers conceived (Kendrick and McEntee, 1967). A number of practitioners have reported low conception rates in heifers bred within a month being vaccinated with IBR vaccine (Roberts, 1971).

Definitive diagnosis is made by demonstrating intranuclear inclusions in the mucosal epithelium, a rise in titer on serology, or by isolating IBR virus (Carbrey et al., 1971). The prognosis is generally favorable as healing spontaneously occurs usually in 10 to 14 days. Occasionally, in severe ulcerative cases in bulls, adhesions may develop between the penis and prepuce. Treatment is not necessary; however, some recommend douching with mild antiseptics (Roberts, 1971). Prevention is the best way to handle IBR infection in beef herds. Vaccination with a modified live virus IBR vaccine is efficaceous if given to non-pregnant females at least 30 days prior to breeding. An intranasal vaccine is available but due to difficulty of administering to adult cattle, it is not recommended.

5) Granular Venereal Disease (GVD): First described by Williams in Switzerland in 1887 as a cause of infertility and abortion (Roberts, 1971). Since then most research has been unproductive but there seems to be little or no current evidence to link it to abortion. It is still regarded as a cause of infertility.

The disease produces a granular vulvovaginitis with elevated granular lesions on the vulvar mucosa that are pink to brownish (Kahrs, 1980). The nodules are submucosal lymphoid accumulations or hyperplastic lymph follicles that may be found in normal cattle. Research revealed that cows with severe granular lesions have a 6 to 10% lower conception rate than non-infected cows (Roberts, 1971).

Although the etiological agent of GVD is unknown, various organisms have been isolated and incriminated. Mycoplasma sp., Corynebacterium pyogenes, Escherichia coli, and others (Roberts, 1971). Experimentally, granular vaginitis has been reproduced by inoculation with Mycoplasma bovigenitalium (Saed and Al-Aubaidi, 1983) and Ureaplasma diversum (Doig et al., 1980). It appears that multiple agents and their combinations induce GVD.

The incubation period of GVD is not known but is thought to be 2 to 3 weeks with lesions persisting for 2 to 4 weeks or longer. The lesions may become chronic or acute lesions may recur. Bulls may also contract GVD with similar lesions appearing on the penis and prepuce.

Treatments are varied and probably any within reason or no treatment at all would be as equally successful (Roberts, 1971). As breeding aggravates GVD, artificial insemination or 2 to 4 weeks of sexual rest are advised.

6) Miscellaneous Conditions: Through the years many other organisms have been isolated from sporadic cases of bovine infertility. However, they may cause similar problems in individual herds as the above mentioned diseases. Viruses isolated and reported to cause infertility include an enterovirus different from the virus causing "Epivag" in Africa but with similar clinical signs, papilloma inducing virus (Roberts, 1971), and bluetongue virus (Luedke and Walton, 1980).

Mycobacteria, Mycoplasma and Mycoplasma-like organisms are also reported to cause infertility. Bovine tuberculosis, caused by Mycobacterium bovis and occasionally M. Avian, is reported to involve the uterus and produce infertility and sterility. Mycoplasma spp. reported to cause repeat breeding include Mycoplasma bovigenitalium, M. agalactiae var. bovis, and M. laidlawii (Roberts, 1971; Saed and Al-Aubaidi, 1983). Ureaplasma sp. cause vaginitis and infertility (Doig et al., 1980) and are commonly found in combination with various bacteria in both bulls and cows (Thornber, 1982).

Bacteria isolated and reported as sporadic causes of infertility may be primary of secondary pathogens and include Pseudomonas aeruginosa (Getty and Ellis, 1967), Haemophilus somnus (Miller et al., 1983), Corynebacterium pyogenes, Streptococcus spp., Staphylococcus aureus, and Bacillus spp. (Thornber, 1982), Escherichia coli, and many others (Roberts, 1971). All organisms produce similar patterns of disease as described previously (vide supra) by infecting both bulls and cows, being venereally transmitted by natural or artificial service.

FETAL PERIOD

Fetal mortality occurs from approximately day 42 of gestation until term. Young and Blair (1974) reported fetal mortality before birth to be 3.7% of PCM. Dennis (1980b) reviewed the literature and reported that 1 to 3% antepartum

deaths appeared to be a "normal" incidence in cows and heifers expected to calve. Many factors during the fetal period influence PCM, mainly environmental and infections. They include intrauterine infections, systemic maternal infections, environmental stresses on the dam, toxins or teratogens ingested by the dam, genetic malformation, abortion, dysmaturity, and premature birth.

1. Intrauterine Infection

Depending on the stage of gestation when infected, intrauterine infections may result in fetal death with resorption, abortion, mummification, or maceration; dysmaturity; premature births; stillbirths; developmental defects; neonatal weakness; birth of viable carriers; or normal viable neonates. The earlier intrauterine infection becomes established in gestation, the more likely the outcome of fetal death and abortion. The later it starts, normal parturition and a surviving neonate is more likely (Dennis, 1980b, 1981). Fetal changes may be caused directly by the organisms or their toxins, or indirectly by placental dysfunction from placentitis. Most fetal deaths result from infection in utero. The main sequellae to intrauterine infection (abortion, dysmaturity, and premature birth) will be reviewed under the fetal period, and developmental defects, neonatal weakness, and viable disease carriers under neonatal period.

2. Abortion

The aborted fetus was described best "as the tip of an iceberg floating in a sea of diagnostic confusion". The confusion resulting from the fact that approximately 65% of aborted fetuses examined fail to yield an etiological diagnosis (Woelffer, 1972; Hubbert et al., 1973; Kirkbride et al., 1973; Dennis, 1969, 1979; Kirkbride, 1977, 1979; KSU, 1981; Jerrett et al., 1984). This combined with the fact that bovine abortion can be an emotional problem often results in frustration of both client and veterinarian (Anon, 1982). Abortion should always be regarded as a herd problem (Dennis, 1979).

The incidence of abortion in a herd is an important indicator of the severity of the problem: 1% is considered "normal" and uneconomical to prevent, 2% a borderline problem, and 3% plus indicates a problem that should be pursued. The causes of abortion are multiple and can result from infectious or non-infectious factors, or by their combinations.

Before any examination is made, it is important to obtain an adequate herd history from the client that should include new additions to the herd, age of dam and previous history, diet, vaccinations, environmental stresses, herd pregnancy rate, and any previous abortions. If the cause of abortion is intrauterine infection, the herd history often reveals other sequellae that comprise the rest of the

abortion complex "iceberg". These include premature births, congenital defects, parturient deaths, birth of weak calves, and neonatal deaths (Dennis, 1980b).

Often a dead calf is found and assumed to be aborted. Unless the calf is undoubtedly of preterminal gestational age, it is important to determine when the calf died: before birth (antepartum death), during birth (partum death), or after birth (postpartum death). This can be done on the basis of gross findings using a modification of the time-of-death (TOD) classification of McFarlane (1965). The fetus and if possible, the placenta should be examined, both are equally important.

1) Non-infectious Abortion: Non-infectious causes of abortion have become an expanding area of research during the past decade. Stuart and Oehme (1982) reviewed environmental factors involved in bovine abortion and Miller (1982) reviewed infectious, nutritional, and environmental causes. Chromosomal anomalies, a major cause of abortion in women, have received little attention in cattle (Carr, 1967). Generally non-infectious factors may be grouped into five categories: nutritional deficiencies, environmental stresses, toxins (plant and chemical), and unknown. Much is to be learned about management to prevent these abortions as they are assumed to comprise a large percentage of undiagnosed abortions. Some known non-infectious causes of bovine abortion are outlined in Fig 2.

2) Infectious Abortion: Miller and Quinn (1975) found that tissues most frequently observed to have lesions of diagnostic significance were eyelids, intestines, liver,

lungs, and placenta. In 68% of cases where the placenta was submitted with the fetus, placental lesions were a major factor in establishing or supporting an etiological diagnosis.

Many surveys have been carried out on bovine abortion and the most commonly diagnosed causes tend to vary from geographic region to region (Table 2). The most common viral causes include IBR, BVD, and PI-3, and the most common bacteria incriminated are C. pyogenes, Leptospira spp., C. fetus ss venerealis, and B. abortus. Aspergillus fumigatus is the most common fungal agent and Anaplasma marginale, Sarcocystis cruzi, and Trichomonas foetus are the common protozoan causes. Bacterial endotoxin has also been found to be a potent abortifacient (Dennis, 1966). Other agents that directly cause abortion and are being diagnosed more with the aid of special testing and cultural techniques are Haemophilus somnus, Chlamydia psittaci, Mycoplasma spp., and bluetongue virus. The various infectious causes of bovine abortion and diseases producing specific or suggestive fetal and placental lesions are summarized in Fig 3. The majority of fetal lesions are nonspecific and laboratory assistance is required for further diagnosis.

Only two organisms are known to produce specific gross fetal lesions; epizootic bovine abortion (EBA) and mycotic infections (Dennis, 1979). Approximately one-third of fetuses aborting from mycotic infection have focal areas of mycotic dermatitis (Hillman, 1969). Mycotic agents can be

demonstrated readily in these areas as well as in sections of eyelid (Miller and Quinn, 1975). EBA produces a striking coarse nodular liver in approximately 30% of infected fetuses (Storz and Whiteman, 1981). More commonly alone or in combination with liver lesions is severe vasculitis with diffuse hemorrhages occurring throughout the body.

Infectious conditions that produce fetal lesions suggestive of diagnosis include IBR, listeriosis, BVD, EBA, and aflatoxicosis. IBR and listeriosis both produce small yellowish, necrotic foci in the liver (Miller, 1982). The necrotic foci may also be found in spleen, kidney, lungs and elsewhere. The foci are larger with listeria infection and small, circumscribed erosions may also be found in the abomasal mucosa of listeria-infected fetuses.

A congested and mottled liver is suggestive of EBA, IBR, chlamydiosis, aflatoxicosis, BVD abortion, and congenital heart defects; it is finer with IBR and BVD infections. BVD is also reported to produce thymic and cerebellar hypoplasia in fetuses infected at specific times during gestation (Miller, 1982). Hydrocephalus may result from genetic and environmental factors but also from in utero infection with BVD or bluetongue viruses. Experimentally PI-3 virus produced interstitial pneumonia in the fetus (Swift, 1973).

Chronic mycotic infections commonly produce a characteristic severe, necrotizing, placentitis and abortion (Hillman, 1969). Caruncles may be found still attached to cotyledons on the fetal membranes dramatizing the severity

of the placentomal lesions. Secondary adventitious placentation is common and the intercotyledonary chorioallantois (ICA) is focally thickened and leathery with extensive superficial necrosis. This is the only placental lesion that is specific for a causative agent (Dennis, 1979).

Leptospirosis, IBR, and EBA produce placental lesions that are similar and suggestive of their cause. All cotyledons are tan to yellowish, atonic, avascular and uniformally affected. The ICA is thickened with serous to brownish gelatinous fluid (Dennis, 1979). Other nonspecific placental lesions indicating infectious abortion include hemorrhages around cotyledons and pericotyledonary areas of the ICA, thickening and opacity of the ICA, and areas of necrosis.

Diagnosis of infectious abortion with the aid of fetal serology has proved to be an effective means for increasing the number of abortions diagnosed (Horner et al., 1973; Sawyer et al., 1973; Miller and Quinn, 1975; Ellis et al., 1978; Moojen et al., 1983). Two and one half times as many aborted fetuses as non-aborted have substantial quantities of immunoglobulins in their fluids (Miller and Quinn, 1975). Antibodies are produced to many agents depending on the antigen and stage of gestation when infected. The fetal calf is generally immunocompetent from mid gestation (Schultz, 1973; Osburn et al., 1982; Butler, 1983). At approximately 100 to 200 days gestation, fetal immunocompetence has been demonstrated to BVD, PI-3, bluetongue,

enteroviruses, B. abortus, Anaplasma and Leptospira spp. Chlamydia, Coxiella burnetti, E. coli and bovine parvovirus produce antibody responses much later at about 250 days gestation (Schultz et al., 1973; Osburn et al., 1982). As more information is gained on interpretating fetal serology, the more significant will its application be (Jerrett et al., 1984). At present, for most serologic tests, any positive fetal titer is significant (Phillips, 1983).

Abortion secondary to maternal systemic involvement can not be overlooked. Any cause of pyrexia such as mastitis or pneumonia may result in abortion by the same mechanism as thermally induced infertility and abortion or by endotoxemia. Maternal hypoxia may result in fetal hypoxia and abortion. Infectious causes of maternal hypoxia include traumatic pericarditis, respiratory disease, and grain overload and acidosis (Miller, 1982).

3. Dysmaturity

Occasionally dead or weak calves are presented at full term with distinct signs of stunting of all or parts of the body; intrauterine growth retardation (IUGR) or dysmaturity is the descriptive term for these calves. Dysmaturity is characterized by neonates with low birth weights, high mortality, and slow growth rates (Coid et al., 1977; Richardson, 1978; Robinson, 1979).

Diagnosis of IUGR can be made on fetuses of accurately known gestational age by comparing their body weights and crown-anus lengths with established standards (Table 3). If the measurements or weights are two standard deviations below the mean of a clinically normal population, they are determined dysmature. The presence of radiopaque lines of the long bones alone confirms IUGR whether the gestational age is known or not. Generally, gestational age is unknown and assuming that there is an absence of radiopaque lines, indicating normal skeletal growth, the chronological age can then be assessed by measuring crown-anus length. A low body weight for this estimated age would suggest IUGR (Richardson, 1978).

Causes of IUGR are relatively unknown. It appears to result from placental dysfunction especially during the last trimester of pregnancy (Dennis, 1981). Done et al. (1980) experimentally inoculated 15 heifers at day 100 gestation with BVD virus; 6 fetuses died in utero and 10 calves were liveborn (one set of twins). Of the 16 calves, 15 had IUGR along with other defects. A marked decrease in weight of the thymus, gastrocnemius muscles, lungs, body weight, tibia and cerebellum were found. Growth arrest lines were also present on the longitudinal sections of some bones. Other causes of IUGR have been proposed but none have been experimentally tested. Postnatal effects of dysmaturity are largely unknown and may be more important than realized (Dennis, 1980a). Diagnosis of IUGR is summarized in Fig 4.

4. Premature Birth

Premature birth is the expulsion before full term of a conceptus capable of independent life (Hubbert et al., 1972). It may be confused clinically with dysmaturity or abortion. An aborted fetus is not capable of independent life. A bovine fetus is capable of independent life from 260 days gestation to term. Differentiation between IUGR and premature birth is made from the fetus being fully developed for its gestational age in a premature birth and stunted with IUGR. Initiation of parturition is primarilly a hormonal process. The fetus determines the length of gestation and dominates the mechanisms initiating it. The bovine uterus is essentially ready for parturition from approximately day 200 gestation till term (Dennis, 1979).

How the fetus determines when to initiate parturition is poorly understood, but the degree of fetal stress is considered to be a major factor. Whatever the cause, the fetal hypothalamus is stimulated to release corticotropin releasing factor that activates the pituitary to release adrenocorticotropin that, in turn, causes the fetal adrenals to release steroids to stimulate fetal prostaglandin release--so called fetal pituitary-adrenal axis. Any hypo- or hyper- function of this mechanism may lead to delayed or premature parturition (Randall, 1978).

Fetal prostaglandins along with placental prostaglandins stimulated by rises in fetal and maternal estrogen levels effect the dam at three important sites: pituitary

gland stimulating oxytocin release; ovaries producing a luteolytic effect and relaxin release; and the uterus along with oxytocin and estrogen increase myometrial activity. Prostaglandins also directly effect the placenta by inhibiting progesterone production and permitting release of relaxin. The outcome of this complex sequence of events is cervical dilatation, increased myometrial activity, and subsequent expulsion of the fetus (Randall, 1978).

NATAL PERIOD

Even though parturient deaths are still fetal deaths (intrapartum), PCM occurring at this time is discussed under the natal period due to significant loss of calves during this short but critical period of life. Grossly, calves dying during parturition can be identified by positive signs of viability but without the first sign of extrauterine survivability, a clot in the umbilical arteries (Dennis, 1979). Even though the umbilical cord has not yet ruptured, evidence of breathing may be found in 14.5 to 80% of calves dying during parturition (Laster and Gregory, 1973; Young and Blair, 1974). Young and Blair (1974) reported that intrapartum fetal deaths comprised 60.5% of the total PCM in their study. The percent of perinatal deaths occurring during birth is variable but can range from 2.5 to 8.6% of all beef calves born (Laster and Gregory, 1973; Young and Blair, 1974; Patterson et al., 1979).

1. Dystocia

Dystocia is defined as difficult birth. Rates of dystocia reported range as high as 83% depending on breed and maternal age (Rice and Wiltbank, 1972; Laster and Gregory, 1973; Laster et al., 1973; Young and Blair, 1974; Philipsson, 1976; Oxender and Adams, 1979; Stables, 1979; Thornberry, 1979). Calf mortality due to dystocia is the largest single source of calf loss (Patterson et al., 1979). Young and Blair (1974) found that 70% of these calves in their study were potentially viable. They based their decision on the presence of localized edema, i.e. a functional heart during birth. Ironically, this is one condition that is almost entirely dependent on management selection and procedures and attention. Dystocia should be one of the easiest causes of loss to make improvements in PCM.

Laster and Gregory (1973) found that dystocic calves had a higher mortality than those not experiencing dystocia--up to four times greater. The cause of fetal death in dystocia is either anoxia from premature rupture of the umbilical cord or, more commonly, varying degrees of birth injury incurred during the difficult or delayed birth.

A combination of management changes have caused an increase in the incidence of bovine dystocia in the past decade: selection for growth rate and size, increased milk production, increased herd size, more intensive management systems, improved nutrition, decreased age at first

parturition, decreased perinatal maternal care and assistance, and seasonal changes. The most significant individual factors influencing parturition are fetal, parental and environmental (Dennis, 1981).

1) Fetal Factors: Birth weight is the most important fetal factor influencing ease of parturition (Rice and Wiltbank, 1972; Philipsson, 1976; Rice, 1979). Other fetal influences include position, presentation, and posture at the time of parturition.

Birth weight is determined by fetal growth rate (genetic and maternal nutrition) and gestation length. Differences among breeds are wide (Philipsson, 1976; Oxender and Adams, 1979; Rice, 1979; Stables, 1979). Maternal nutrition has little effect on birth weight except severe undernutrition during the last 4 to 8 weeks of gestation.

Dystocia increases an average of 2.3% for each kg. increase in birth weight (Dennis, 1981). However, mortality is not limited only to heavier calves. Relatively high percentage of both extremely light and heavy calves are born dead (Donald, 1963; Rice, 1979).

Roy (1980) described an index (Z) to estimate the percentage of calving dificulties likely to occur in any particular situation:

$$Z = \log BW - 0.40 \log DW - 0.10P$$

where BW is the birth weight of the calf (lbs.); DW is dam's weight (lbs.); P is parity of the dam with 0 for the first parity, etc.

Laster and Gregory (1973) found that death losses from all types of parturition were 3.8% higher in purebred lines of cattle than in reciprocal crossbred calves. Also, when calving assistance was required, perinatal and early post-natal calf mortality was 11.6% higher in purebred versus crossbred calves. This suggested that crossbred calves could tolerate the higher levels of stress associated with difficult birth better than purebreds. Inbreeding has also been shown to increase PCM.

Sex of the calf significantly affects mortality during birth. The frequency of dystocia with male calves, approximately 3 kg. heavier than females, is up to twice that of female calves (Patterson et al., 1979; Dennis, 1981), and calf losses were higher in males than females (Laster and Gregory, 1973).

2) Parental Factors: Age at first parturition is important as the incidence of dystocia is higher in heifers less than 2 years of age even though birth weights were lower than in older cows (Lindhe, 1966; Laster and Gregory, 1973). Parity of the dam, directly related to age, is also important as the incidence of dystocia is 2 to 4 times higher in heifers than cows (Rice and Wiltbank, 1972; Philipsson, 1976). These two factors combined, first parity in heifers less than 2 years old, produce the major maternal factors influencing dystocia and subsequent PCM.

The age and parity difference in dystocia is apparently due to the smaller pelvic opening in 2-year-old heifers than in older cows. Rice and Wiltbank (1972) reported dystocia rates of 28% for heifers with pelvic areas less than 200 sq.cm. Small pelvic area and large birth weight in combination is by far the most lethal of all factors influencing difficulty of birth and calf survivability-- the feto-pelvic relationship. An incompatability in this relationship is what should be avoided when formulating management and breeding strategies to decrease the incidence of dystocia in first calf heifers.

In multiparous females, the incidence of dystocia is significantly lower but there are instances where it can be a problem. A proportion of cows with dystocia suffer from uterine inertia (Young and Blair, 1974). This can be due to imbalances of trace minerals in the diet or a lack of protein and energy. Uterine inertia can also result from exhaustion from prolonged uterine contractions to deliver an oversized or malpositioned fetus (Hindson, 1978). Vulvar stenosis has also been reported as a cause of dystocia in Jersey cattle and to a lesser extent, in Angus and Friesian cows (Leipold and Dennis, 1980).

The sire of the calf, as well as the maternal grandsire, affect the incidence of dystocia and PCM primarily through fetal birth weight (Philipsson et al., 1979). With the advent of crossbreeding with larger exotic breeds, the breed of the sire has become a significant cause of dystocia

in both cows and heifers. Dystocia increases with breed size. Charolais, Simmental and Limousin bulls sire fetuses with higher dystocia and PCM rates. Frequency of dystocia in British breed cows is 2 to 3 times that in herds using larger-sized sires than in those using Hereford and Angus sires (Tayler, 1976).

Various researchers have effectively reduced herd dystocia rates by selecting for sires with low dystocia rates. Successfull attempts have also been made to arrive at breeding values and heritability estimates for PCM by reducing dystocia (Berg, 1979; Bar-Anan, 1979).

3) Environmental Factors: Seasonal influences on dystocia can occur directly as is seen with variations of birth weight as fall-born calves are, on the average, one half pound lighter than spring-born calves. Season can influence the energy intake of the dam predisposing to uterine inertia and dystocia. In either case, winter calving is usually more difficult and causes a higher stillborn rate than calving during other seasons (Martin et al., 1975a,b; Philipsson, 1976).

2. Birth Trauma

Birth trauma commonly occurs during dystocia and it may be so severe that the fetus dies. However, if trauma is sublethal, the injured calf may die within a few hours of birth and be classified as neonatal weakness. Laster and Gregory (1973) found that calf losses in two-year-old

heifers with no dystocia was higher than in older cows. They proposed that calves from two-year-old heifers that had no assistance may have needed assistance and that the stress and injury of the calf from prolonged parturition increased mortality.

Meningeal hemorrhages are important manifestations of injury to the CNS arising from trauma and/or hypoxia during birth (Haughey, 1975). CNS hemorrhages were found in calves dying during and immediately after parturition. Calves from assisted difficult births had a high incidence of meningeal hemorrhages. The severity of hemorrhage is proportional to the amount of traction applied during assistance. Severe injury from assisted or unassisted births lead to death during or imediately after birth, less severe injury impairs physical and behavioral activity and thus prejudices neonatal survival, and more subtle injury may lead to survivors becoming handicapped later in life (Haughey, 1975).

Hindson (1978) developed an objective formula for use with dystocia to assess the traction ratio and best method of extraction:

$$\text{Traction Ratio} = II/DD \times P1/P2 \times 1/E$$

where II = inter-ischial distance, DD = digital diameter at the fetlock joint, P1 = parity factor of 0.95 for heifers, P2 = correction factor for posterior presentation of 1.05, E = exotic correction factor of 1.05 for breeds known to have muscular hypertrophy. From experimental studies, the following tentative correlations were formulated:

Ratio above 2.5 = oversize not present

Ratio 2.3 - 2.5 = oversize possible but traction likely to be successful

Ratio 2.1 - 2.3 = oversize, traction difficult, surgery should be considered

Ratio below 2.1 = oversize, live calf by traction improbable, surgery required

The force exerted on the fetus can vary widely from approximately 65 kg. during natural calving to greater than 5,000 kg. under mechanical traction (Hindson, 1978). The amount of force (kg.) exerted on the fetus for different types of traction and examples of the force for the types of traction are depicted in Fig 5. It is recommended that, when delivery can not be accomplished with maximal manual assistance or sub-maximal assistance with a calf puller, surgical extraction should be performed.

Other reported types of birth trauma are numerous but commonly include femoral nerve injury and paralysis; ruptured quadriceps muscles; stifle luxation; focal hemorrhage in the cerebrum and cerebellum; hepatic rupture; unilateral hemothorax; separation of costochondral junctions; fractured ribs; discontinuity of the vertebral column; local edema of head, limbs, and tongue; fracture of humerus; slipped capital femoral epiphysis; intramuscular hemorrhage; and hemorrhage in and around the stifle, the hip joints, intervertebral fibrocartilage, and axillae (Tryphonas et al., 1974; Haughey, 1975; Gopal, 1977; Hamilton et al., 1978).

NEONATAL PERIOD

Most neonatal deaths occur during the first seven days of life (Oxender et al., 1973; Speicher and Hepp, 1973; Young and Blair, 1974; Martin et al., 1975a,b). PCM occurring during the neonatal period is reported to range from 2.0 to 4.2% of all females expected to calve, with an average incidence of approximately 3%. Young and Blair (1974) using systematic classification for TOD reported that neonatal deaths in their study comprised 35.8% of the PCM complex. Patterson et al. (1979) found that these deaths comprised 62% of the PCM complex, with 75.1% dying during the first seven days. Others have reported neonatal deaths accounting for 64 to 82% of the PCM, with most occurring during the first two weeks (Martin et al., 1975a,b; Roy, 1980).

1. Adaptation

The first one to six hours of a calf's life is critical for its survival (Dennis, 1981). The postpartum period begins when the umbilical cord is severed and the calf must provide for its own life support. Many times the calf will take its first breath and continue to breath before the umbilical cord is broken, sometimes even while still in the birth canal. However, as long as the umbilical cord is intact, the calf does not have to continue the adaptation process. As soon as it ruptures, the calf is forced to

adapt or die. That is why severence of the umbilical cord instead of initiation of respiration is the physiological beginning of the adaptive period.

After the umbilical cord ruptures, adaptation normally occurs in the following sequence: respiration, circulation, thermoregualtion, mobility, nutrition and microbial defense. To advance in this critical sequence, the calf must effectively adapt to each physiological change preceding it. Randall (1978) presented an excellent review of perinatal adaptation.

1) Respiration: For survival, the neonate must quickly overcome the stress of parturition and reverse the hypoxia resulting from umbilical cord severance. The factors initiating respiration are both chemical and physical. Stimulation of the respiratory center by a build-up of CO_2 during parturition acts chemically to initiate breathing along with the mild acidosis. Physically, there is an elastic recoil of the thorax after its compression through the birth canal (Head's paradoxical reflex). This and the mild asphyxia, are responsible for lung expansion and intitiation of the first breath. Continued respiration is stimulated by tactile stimuli, cooling, and the rise in blood CO_2 level (Randall, 1978).

2) Circulation: The calf must then convert from a fetal cardiovascular pathway to an adult pathway. For this to happen, the umbilical arteries must seal off, the foramen ovale must close, and the ductus arterious constrict. In a

neonate with a functional heart, the umbilical arteries have an elastic property so that as the umbilicus is severed, they recoil and seal the ruptured ends and the blood clots within them (Randall, 1978).

When placental blood pressure is lost, the pressure in the right side of the heart drops and the pressure exerted by the left side increases. This increased left atrial pressure mechanically closes the foramen ovale. Last to functionally close is the ductus arteriosus, stimulated by the increase in oxygen tension of the blood; complete closure may be delayed for two weeks or more and it is important to note that until complete closure is accomplished, it can reopen with a decrease in oxygen tension (Randall, 1978).

3) Thermoregulation: The neonate must adapt to the temperature of its surroundings and maintain its own body heat. Newborn calves are especially susceptible to hypothermia immediately after birth when they are wet with amniotic fluid. The large surface area to body weight ratio makes it more difficult for neonates than for larger, better insulated adults to maintain body temperature (Dennis, 1981).

They accomplish thermoregulation by two ways; nonshivering and shivering thermogenesis. Nonshivering is accomplished through the metabolism of energy-rich brown fat present mostly around the kidneys, mesentery, pericardium and epicardium at birth. The calf is born with enough brown

fat energy stores to keep it going without appreciable maternal nutrition for 3 days in mild weather. Shivering thermogenesis is not obvious in moderately cold conditions but becomes obvious as the severity of cold exposure increases. This form does not occur until nonshivering thermogenesis is at its fullest potential (Alexander, 1975).

4) Mobility: Mobility also helps in maintaining body temperature but more importantly, it is essential for survival and continued adaptation. Calves are usually standing within 30 minutes after birth and are walking soon after. If it takes a calf longer than one hour to stand, it should be considered potentially abnormal (Dennis, 1984). Upon standing, the calf nurses and further establishes the neonatal-maternal bond. Failure of or delay in forming this bond is detrimental for the calf and potentially costly to the producer.

5) Nutrition and Microbial Defense: This step is dependent on cooperation of the dam as well as the complex suckling reflex (thigmotaxis) occurring between the calf and its dam. By obtaining colostrum from its dam, the calf has overcome the last two steps in adaptation. It has acquired nutrition and has also ingested maternal antibodies contained in colostrum (Dennis, 1984).

It is important that the calf obtains passive immunity because even though it can respond to some antigens at birth, it is not functionally immunocompetent. From the stress of birth and adaptation, the calf has high levels of

circulating corticosteroids that inhibit its inherent defenses and place it at risk from agammaglobulinemia, lymphopenia, and decreased phagocytic activity (Osburn et al., 1974). The calf must ingest antibodies within the first 24 hours after birth for them to be absorbed. Maximum absorption occurs during the first six to eight hours of life (Dennis, 1981).

At birth, the intestinal tract is sterile but microbial colonization is relatively complete by seven days. The first milk not only helps in colonization but also provides protective local and systemic antibodies for the neonate to deal with toxic by-products (endotoxins and enterotoxins) from the multiplying bacteria. Development of immunologic responsiveness and an effective mucosal barrier against penetration by pathogenic agents is an important adaptation process. The potential growth of a calf is positively correlated with its globulin level at the end of the first week of life (Thornberry, 1979) and calfhood disease and mortality is directly related to failure of maternal antibody transfer (McGuire et al., 1976).

2. Developmental Defects

Congenital or developmental defects can result from genetic, environmental, toxic, and infectious factors occurring during gestation. The incidence of congenital abnormalities is reported to range from 0.2 to 3% of all calves born, with an average of 0.5% (Laster and Gregory,

1973; Leipold, 1978, 1980). Of defective calves reported, 84% die from neonatal asphyxia with 9.1% having evidence of infection (Leipold, 1978).

Before day 14, the developing zygote is susceptible to genetic mutations and chromosomal aberrations but is resistant to teratogens. Experimental infections with viruses (bovine leukemia, bluetongue, IBR, BVD, bovine parvovirus, and Akabane) all failed to effect 7 to 10 day-old embryos (Archbald and Godke, 1984). During the embryonic period (day 14 to 42), the embryo is highly susceptible to teratogens but susceptibility decreases with embryonic age as the various critical periods of organogenesis pass. The fetus (day 42 plus) becomes increasingly resistant to teratogenic agents with age except for later developing structures such as cerebellum (day 150), palate and urogenital system (Leipold and Dennis, 1980).

Congenital defects are usually classified by the organ system primarily involved: central nervous system, skeletal, visceral, cardiac, and multiple. Gopal (1977) found that visceral and cardiac defects comprised 63.5% of all bovine defects examined in his study, skeletal defects 18.8%, and C.N.S. 17.8%.

Teratogenic environmental factors identified in cattle include: toxic plants, drugs, trace elements, and irradiation (Leipold, 1980). Crippled-calf disease in Utah and Kodiak Island has been linked to ingesting lupines between 40 to 60 days of gestation. Other plants suspected

of causing deformities in calves include Senecio, Indigofera spicata, Cycadales, Blighia, Loco plants, Papaveracea, Vinca, tobacco and related plants (Keeler, 1978). Rectal palpation between days 35 to 40 gestation may cause atresia coli (Leipold et al., 1983).

Viruses are the most important infectious agents causing developmental defects (Done, 1978; Leipold et al., 1983). Cerebellar atrophy is caused by prenatal infection with BVD virus, hydranencephaly by bluetongue virus, and Akabane virus has caused hydranencephaly and arthrogryposis.

Neonatal bovine genetic defects of concern in the United States include hypotrichosis in Hereford cattle, arthrogryposis, hydrocephalus, mannosidosis in Angus cattle, osteopetrosis, tibial hemimelia, polydactyly, and syndactyly (Leipold et al., 1983).

Differentiation between genetic defects and environmental or infectious induced defects is important but often difficult to do. In general, environmentally caused defects follow a seasonal pattern and known stressful conditions, they may be linked to maternal disease rather than following a familial pattern (Leipold, 1980). Maternal disease patterns vary but calves from heifers are more frequently affected, fetal Ig levels are frequently detectable, abortion incidence is increased, and morbidity may be observed in the herd or other associated animals (Leipold, 1980).

Most genetic defects are simple autosomal recessives and follow a Mendelian pattern of inheritance. Other inheritance patterns described are overdominant, dominant, incompletely dominant, sex linked, and polygenic (Leipold, 1980). Done (1978) listed some differential characteristics of developmental defects in cattle caused by mutant genes and viral teratogens.

3. Neonatal Weakness

Neonates surviving parturition but clinically weak, usually unable to rise or can walk only a short distance, and usually die within minutes to hours after birth. The calves can be identified grossly by a clot in the umbilical arteries and by various degrees of lung aeration. The best description for these calves is neonatal weakness (NNW). NNW may be due to a multiplicity of non-infectious, infectious, and undetermined factors or their interactions. It is a major cause of PCM during the first 24 hours of life (Dennis, 1980a), and are often reported as stillbirths. Factors predisposing to NNW are many and are summarized in Fig 1.

1) Non-infectious Neonatal Weakness: Non-infectious causes of neonatal weakness include: fetal stress, birth trauma, genetic factors, and exposure. Fetal stress from hypoxia and acidosis frequently result from difficult or prolonged birth especially in first-calf heifers. Hypoxia in utero may result in meconium staining of the fetal hair

coat. Decrease in oxygen tension of fetal blood causes a relaxation of the anal sphincter, hyperperistalsis of the intestines, and voiding of meconium into the amniotic fluid. If hypoxia continues, the fetus attempts respiration and may aspirate amniotic fluid and meconium into its lungs. Survival of such calves may be improved by prompt experienced assistance at and immediately after birth (Dennis, 1981).

Trauma from a difficult and prolonged birth has been discussed under dystocia. It is sublethal forms of birth trauma that predispose to NNW. Meningeal hemorrhages and other CNS injuries play a major role in failure of calves to adapt (Haughey, 1975). It is reasonable to conclude that birth trauma and hypoxia from fetal stress are the two major causes of NNW.

Data is available that points to the existence of genetic factors that affect calf survival for the first few hours of life and later (Thornberry, 1979). Crossbred calves seem to tolerate higher levels of parturient stress than purebreds (Laster and Gregory, 1973), and inbred calves tend to be weaker and less vigorous (Woodward and Clark, 1959). The genetic make-up of the calf also determines, to a large extent, its birth weight. The influence of light and heavy birth weights on calf mortality have been discussed (vide supra).

Environmental stress from exposure to excessive wind, wetness, and cold temperatures has adverse effects on young calves. Severe cold stress, sufficient to cause hypothermia in calves, also produces severe subcutaneous hemorrhage and edema, especially of the extremities (Olson et al., 1980a), and delayed absorption of colostral immunoglobulins (Olson et al., 1980b). Clinical weakness of calves resulting from cold exposure has been experimentally reproduced (Olson et al., 1981).

Necropsy findings in calves dying from uncomplicated exposure include normal hydration, diffuse subcutaneous edema of distal limbs and tail, fat metabolism, dark congested muscles, peripheral edema, and hemorrhages below and around the carpus and hocks (Young and Blair, 1974; Dennis, 1980a; Olson et al., 1980a, 1981).

2) Infectious Neonatal Weakness: Information on infectious causes of NNW is limited. Most infectious conditions do not result in death till after the first week of life. Calves dying then exhibit gross signs of partial or complete metabolisum of their body fat stores, indicating starvation complicated by an infectious agent. The majority of neonatal infections will be discussed under infectious causes of complicated starvation, congenital infections, and a NNW syndrome called Weak Calf Syndrome.

3) Congenital Infections: Neonates sublethally infected in utero early in gestation may become tolerant to that infectious agent and not produce antibodies to combat it.

This allows the agent to multiply unchecked. If the neonate is infected later in gestation, the fetus can respond immunologically and attempt to produce antibodies to combat the infection but due to the ineffectiveness of its immune system, it is soon overwhelmed by infection. These neonates are sometimes born alive and are carriers or shedders of that infectious agent (Luedke et al., 1977; Done et al., 1980). The calves are usually weak, listless, have a high mortality, poor growth, and usually develop clinical signs of disease soon after birth.

Maternal infections resulting in fetal infection and NNW can be bacterial, viral, or protazoan. Agents that cause NNW include B. abortus, M. bovis, H. somnus, Mycoplasma sp., Chlamydia psittaci (Stauber, 1976; Roy, 1980) and viruses such as BVD, bluetongue, adenoviruses, malignant catarrhal fever, and bovine parvovirus (Plowright et al., 1972; Dierks et al., 1976; Luedke et al., 1977; Done et al., 1980; Storz and Young, 1980). Zaugg and Kuttler (1984) experimentally demonstrated that maternal Anaplasma marginale infection can cause NNW.

4) Weak Calf Syndrome: In the past two decades, NNW has been brought to the attention of veterinarians in certain geographic regions as a substantial and frustrating part of the PCM complex. During this time, the clinical term weak calf syndrome (WCS) was coined (Dennis, 1980a). The term is useful for describing the problem to laymen but its misusage seems to have slowed progress in the search for the various

causes of NNW. It has also hindered understanding of the condition. WCS is commonly used to lump together all forms of NNW; infectious, non-infectious, and undetermined. It is suggested that the term WCS be left as a descriptive term for laymen and that veterinarians use terminology like neonatal weakness to be more exact for the cause of WCS.

The syndrome was reported in one study to account for 6.95% of all calf deaths (Khera, 1981). WCS is characterized by the same criteria as found with intrauterine infections, late abortions, stillbirths, and weak calves with a high mortality and poor growth rate (Card et al., 1974; Stauber, 1976). The syndrome is more common in calves from first-calf heifers or from new additions to the herd. Usually only one affected calf is reported per cow (Rice, 1979).

Gross findings of calves with WCS often reveal lesions characteristic of various infectious and non-infectious causes. Necropsy findings include hemorrhage and edema of the subcutaneous tissues of the extremities, especially around the carpal and tarsal joints and extending distally (the most common finding); polyarthritis with hemorrhagic synovial fluid containing various amounts of fibrin; congestion and hemorrhage of the abomasal mucosa with some erosion and ulceration; and various degrees of body fat metabolism (Card et al., 1974; Dierks et al., 1976; Stauber, 1976).

Causative factors incriminated in WCS include immunosuppressive viremia, BVD, bluetongue virus, adenoviruses, Mycoplasma sp., C. psittaci, H. somnus, low maternal nutrition during the last month of gestation, and exposure (Dierks et al., 1976; Stauber, 1976; Rice, 1979; Kvasnicka, 1984). Olson et al. (1981) found that lesions associated with exposure were similar to those reported for WCS and the lesions appeared more frequently in calves from dams fed low-protein diets than calves from dams fed adequate-protein diets.

4. Uncomplicated Starvation

Starvation is a major cause of perinatal lamb mortality. Dennis (1974) reported that 46.7% of all perinatal lamb losses in his study were due to uncomplicated starvation. These lambs were easy prey for predators. In beef cattle, uncomplicated starvation does not have as big an impact on PCM as it does in lambs. The stress of starvation may lead to increased susceptibility to various pathogens with the calf then dying of complicated starvation.

Young and Blair (1974) found that 9.8% of the PCM in their study died from starvation. Many of the calves were known to be twins or calves left motherless after their dam died from metabolic or other diseases. Their finding of a

fairly high incidence of PCM from starvation appears to be related to the extensive husbandry practiced in the region of their study.

Calves from first-calf heifers tend to be victims of starvation more frequently than calves from multiparous cows. This may be due to two factors: failure of a strong maternal-neonatal bond resulting in desertion by the dam, and secondly, difficult birth with a calf too weak or injured to stand and nurse.

Lack of nourishment makes calves more susceptible to chilling, hypothermia, and death from exposure. However, the stress of starvation does not appear to be a contributing cause of the peripheral edema observed in these deaths (Young and Blair, 1974).

5. Disease Complicated Starvation

Late in the neonatal period (day 8 to 28), starvation complicated by various disease conditions is the major cause of PCM. Gross observations reveal partial to complete metabolism of body fat and significant lesions in other parts of the body such as digestive tract, lungs, central nervous system, and skeletal system individually or in combination.

Neonatal weakness and congenital defects can predispose to infection and starvation. The effects of mild starvation and infection are additive and interrelated. Infection can result in starvation and starvation can result in increased

susceptibility to pathogens. The end result is the same, the calf uses up its body stores and is overwhelmed by infection. Death results from disease complicated starvation

Major infectious conditions complicating starvation in beef calves are enteritis, pneumonia, and septicemia. A wide range of bacterial and viral organisms has been incriminated in neonatal infections. The susceptibility of calves to these infections is directly related to prompt ingestion and effective absorption of colostral immunoglobulins. Responsiveness to treatment and prognosis is also dependent on effective passive transfer of maternal immunoglobulins. Antibiotics and chemotheraputic agents are usually ineffective in preventing death in agammaglobulinemic calves (Dennis, 1981).

1) Failure of Passive Antibody Transfer: Immunogenesis is suppressed in the neonate for 10 to 14 days by the high level of coricosteroids during and after birth (Osburn et al., 1974). Immunological resistance to pathogens is almost entirely dependent on absorption of colostral antibodies, therefore failure of passive transfer of maternal immunoglobulins (Ig's) is directly related to calfhood disease (Boyd, 1972; McGuire et al., 1976; Pfeiffer and McGuire, 1977; Dardillat et al., 1978; Lumba et al., 1978; Davidson et al., 1981).

The potential growth of a calf is positively correlated with its globulin level at eight days of age (Thornberry, 1979). The concentration of Ig's reaching a calf's blood

stream is dependent on the quality and quantity of colostrum; how soon and how frequently after birth it is ingested, how it was ingested, some environmental factors, and unknown influences.

It is important that a calf receive 2 liters (5% of its body weight) of its dam's colostrum immediately after birth (Drost, 1980). The rate of Ig absorption from the intestines declines from birth to no absorption at approximately 24 hours (Dennis, 1980). There is a lag of about 5 hours between suckling and 50% maximum serum antibody levels in calves (Logan et al., 1978). One feeding does not provide sufficient Ig protection.

If colostrum must be administered to the calf, feeding in the presence of the dam significantly increases Ig absorption (Drost, 1980). One study found the mortality rate to one month of age for dairy calves suckling colostrum was 4% compared with 9% for calves given colostrum from a bucket (Roy, 1980). Colostrum can be stored at -18 to -25 C for at least 6 months without reducing its protective value provided it is frozen in amounts less than 0.6 liters (E.E.C., 1979). If colostrum is not available, a formula recommended that can assist in protecting the neonatal calf consists of a whipped egg in 0.3 liters of watter with one half teaspoon of castor oil and 0.6 liters of whole milk (Roy, 1980). It should be given three times a day for the first three to four days of life. The rationale for this treatment may lie in the fact that egg white has a marked

antibacterial action on certain strains of E. coli, and that egg albumin, like globulins of colostrum, can pass unchanged into the blood stream of the calf during the first 24 hours of life. Experimentally, one whole egg given daily for the first 10 days of life to calves deprived of colostrum had a protective action against septicemic E. coli infection (Roy, 1980).

There are seasonal variations in passive transfer of Ig's to newborn calves. Gay et al. (1983) found the mean monthly serum IgG_1 concentrations were lowest in winter, increased during spring and early summer to reach their peak in September, and then decreased. Olson et al. (1980b) discovered that experimentally-induced hypothermia delayed Ig absorption.

Nutrition of the dam is reported to influence passive transfer of Ig's. Blecha et al. (1981) restricted the protein intake of pregnant heifers during the last 100 days of gestation and found a selective decrease in absorption of IgG_1 and IgG_2 in their calves. This was contrary to earlier work (Olson et al., 1980b).

Other effects on passive transfer of maternal Ig's include genetic, parturient, and unknown factors. Muggli et al. (1983) found that a selection line of calves was one of the most important factors influencing Ig concentration. Also in this study, increased calving difficulty was associated with a decrease in IgG_1 levels of the calves. Sex of calves had no effect on Ig levels.

Recently fetal and neonatal hormonal influences on absorption of Ig's have been studied. Higher 24 hour serum cortisol levels were negatively correlated to 48 hour Ig concentrations and positively correlated to calf mortality in one study (Scott, 1980). However, the cortisol levels may have only been in response to heat stresses placed on the calves. Other studies found that high cortisol levels in calves at birth did not interfere with Ig absorption (Johnston and Oxender, 1979). Also, exogenous corticosteroids used for inducing parturition apparently do not hinder absorption, but early calving may reduce the volume of colostrum available and the amount ingested (Hoerlein and Jones, 1977).

Thyroid activity of the newborn related to levels of serum Ig's and health have also been studied (Cabello and Lavieux, 1978). Triiodothyroxin (T3) and the free thyroxin index at birth were significantly lower in healthy calves than in those suffering from diarrhea. Levels of plasma hormonal iodine at birth and plasma globulins at 48 hours were negatively correlated. Boyd and Hogg (1981) found no significant correlation between thyroxin (T4) and 24 hour Ig concentration.

Both cortisol and thyroid hormone have induced premature closure of the intestinal epithelium to globulins in species other than cattle (Chan et al., 1973; Cabello et al., 1980).

Despite adequate colostrum intake early in life approximately 20 to 30% of neonatal calves are reported to be hypogammaglobulinemic (Osburn et al., 1974). The reasons are unknown but genetic, environmental, and hormonal factors previously described may be significant.

2) Enteritis: The major calf disease in beef cattle continues to be enteritis (Oxender and Adams, 1979). Young and Blair (1974) reported that enteritis/gastroenteritis accounted for 12% of the total PCM in their study. This was second only to parturient deaths. Radostits and Acres (1974) reported that 51.8% of newborn calf problems were intestinal. The problem was twice as great in calves under two weeks of age compared to older calves (Oxender and Adams, 1979).

There are three current etiologic theories on neonatal calf scours: infection by enteropathogenic bacteria and viruses; hypogammaglobulinemia due to failure of passive transfer of maternal Ig's; and congenital immunodeficiency. Most likely there is a combination of the three (Larvor, 1979). Colostrum fed soon after birth provides protection from coli-septicemia but does not completely prevent colibacillary diarrhea.

Neonatal diarrhea leads to severe dehydration and metabolic acidosis within hours and complicated starvation develops within a few days even with aggressive treatment. This results from malabsorption commonly associated with persistent diarrhea (Moon, 1978). The acidosis and

dehydration are primary clinical problems and the most beneficial treatment is to replace the fluids and correct the acidosis (Oxender and Adams, 1979).

Enteropathogenic bacteria and viruses can affect neonates independently or in combination (Mebus et al., 1977; House, 1978). The majority of diarrheic epizootics are associated with multiple enteropathogens. The main nutritional factor affecting the incidence of diarrhea is feeding milk substitute diets containing "severely" pre-heated spray-dried skim milk powder or non-milk proteins. The importance of adequate ingestion of colostrum cannot be overemphasized. Antibiotics and chemotherapeutic agents are ineffective in preventing mortality in agammaglobulinemic calves. Serum samples of diarrheic calves should be taken and tested for Ig concentration to assess the prognosis and potential responsiveness for treatment.

Enterotoxic E. coli appears to be the commonest single agent causing diarrhea (Acres et al., 1975; Mills and Tietze, 1984). E. coli can be a primary pathogen or secondary associated with viruses and protozoa, or it can exert its enterotoxogenic properties. E. coli infection commonly enters through the navel and becomes septicemic.

Rotavirus and coronavirus are major causes of neonatal diarrhea (Cimprich, 1981). Rotavirus effects calves 5 to 10 days of age and coronavirus between 5 and 21 days. Protective antibodies to rota and corona viruses are not circulating IgG but local IgA found in colostrum and milk.

Both viruses effect the villous tips causing atrophy and the lesions are more severe with corona virus (Cimprich, 1981). Rotavirus is extremely resistant to environmental factors and may survive for several years in buildings and cause annual outbreaks on some farms (Dennis, 1981).

Torres-Medina (1984) infected gnotobiotic calves with combinations of calf rotavirus (CRV), enterotoxogenic E. coli (ETEC). and non-enterotoxogenic E. coli (NETEC). Severe diarrhea and villous atrophy were observed in all calves dually infected with CVR and either ETEC or NETEC. Calves infected with CRV or ETEC only also had severe diarrhea and villous atrophy. Dual CRV - ETEC infection produced the most severe lesions and death occurred as well as in the ETEC group. This study provided much insight into the interaction and pathogenicity of rotavirus and E. coli in neonatal bovine diarrhea.

Although coronavirus, rotavirus, and E. coli are the major infectious agents that should be considered first in diagnosing neonatal calf diarrhea, several other diseases should also be considered; Salmonella spp., Chlamydia sp., and Clostridium perfringens are potent causes of neonatal diarrhea. The type C toxin produced by C. perfringens can be acutely fatal to neonates and diarrhea is not often observed. The usual history is a healthy calf out of a heavy milking dam that died acutely and bloated soon after death.

Although viruses other than rota and corona viruses are not frequently encountered, BVD, IBR, bovine parvovirus, and some unidentified viruses may be incriminated (Evermann, 1979; Cimprich, 1981). BVD and IBR viruses are also associated with respiratory infections in neonatal calves.

Occasionally, the protozoan Cryptosporidia causes diarrhea in neonatal calves but it is usually associated with poor management. Some reports indicate that it may be found in combination with E. coli.

Recently preventive vaccines for pregnant dams have been developed. They function by stimulating higher maternal concentrations of colostral antibodies to the various enteropathogens. When the calf ingests the specific antibody-rich colostrum it is better equipped to fight off invading pathogens. With this approach, neonatal calf scours has been significantly reduced and the responsiveness of affected calves to treatment has increased (Oxender and Adams, 1979; Kvasnicka, 1984).

3) Miscellaneous Infections: In beef cattle, except for enteritis, the other neonatal infections during the first week of life tend to be septicemic or toxemic. As these infections progress they localize in various organs such as joints, brain, lung, kidney, liver and intestines. Transmission of infection may be by contamination of the umbilical cord, inhalation or ingestion (Dennis, 1980a,b). There are many predisposing factors to these septicemias but the most common is hypogammaglobulinemia. Septicemic or

toxemic conditions are usually associated with one or more of the following: enteritis, pneumonia, peritonitis, pleuritis, polyarthritis, omphalophlebitis, meningitis, encephalitis, and focal suppurative lesions in various organs, especially kidneys and liver.

Many bacteria have been isolated from septicemia in neonatal calves including hemolytic and nonhemolytic E. coli, Pasteurella spp., Fusobacterium necrophorum, C. pyogenes, Salmonella spp., Streptococcus spp., and Staphylococcus aureus (Nat. Acad. Sci., 1968; Young and Blair, 1974; Davidson et al., 1981).

In dairy calves pneumonia as well as enteritis is a major disease (Oxender et al., 1973). Evermann (1979) reported that in the northwestern U.S., pneumonia represented 19% of the total beef and dairy calf losses combined. Pneumonia in beef calves is normally not a major problem (Young and Blair, 1974). IBR, PI-3, and BVD were the most commonly isolated viruses associated with calfhood respiratory diseases. Other less commonly found viruses included bovine adenoviruses, non-IBR herpes virus, and syncitial virus (Evermann, 1979). BVD virus can result in pneumonia, diarrhea, pneumo-enteritis, and neonatal weakness (Evermann, 1979). It is frequently present in affected respiratory tract tissues together with other potential pathogens such as PI-3, IBR, and P. hemolytica. A synergism may occur and experimentally it impaired the calf's ability to clear IBR virus from its lungs (Potgieter et al., 1984).

Evermann (1979) found that 48% of viruses isolated in septicemic cases of pneumo-enteritis cases were unidentified. The predominant isolates were tentatively characterized as non-bovine enterovirus picornaviruses.

REFERENCES

Abbitt B. Trichomoniasis in cattle. In: Morrow DA, ed. Current Therapy in Theriogenology. Philadelphia: WB Saunders Co, 1980;482-488.

Acres SD, Laing CJ, Saunders JR, Radostits OM. Acute undifferentiated neonatal diarrhea in beef calves. 1. Occurrence and distribution of infectious agents. Can J Comp Med 1975;39:116-132.

Alexander G. Body temperature control in mammalian young. Brit Med Bull 1975;31:62-67.

Anonymous. The enigma of the abortus fetus. Illinois Cont. Educ. Letter, 1982;6-9.

Archbald LF, Godke RA. What are the risks for transmission of disease with embryo transplantation. Proc Ann Conf AI Emb Trans Beef Cattle, Denver, January 14, 1984; 41-46.

Baker AL, Queensberry JR. Fertility of range beef cattle. J Anim Sci 1944;3:78.

Bar-Anan R. A breeding strategy for reducing perinatal calf mortality in heifer calves. In: Calving Problems and Early Viability of the Calf. Martinus Nijhoff Publ, The Hague, 1979;149-158.

Berg RT. Breeding considerations for minimizing difficult calving. In: Calving Problems and Early Viability of the Calf. Martinus Nijhoff Publ, The Hague, 1979;133-140.

Blecha F, Bull RC, Olson DP, Ross RH, Curtis S. Effects of prepartum protein restriction in the beef cow on immunoglobulin content in blood and colostral whey and subsequent immunoglobin absorption by the neonatal calf. J Anim Sci 1981;53:1174-1180.

Boyd JW. The relationship between serum immune globulin deficiency and disease in calves: a farm survey. Vet Rec 1972;90:645-649.

Boyd JW, Hogg RA. Field investigations on colostrum composition and serum thyroxine, cortisol and immunoglobulins in naturally suckled dairy calves. J Comp Path 1981;91:193.

Butler JE. Bovine Immunoglobulins: An augmented review. In: Vet Immunology and Immunopathology Elsevier Science Publishers, Amsterdam, 1983;4:43-152.

Cabello G, Levieux D: The effects of thyroxine and climatic factors on colostral gammaglobulin absorption in newborn calves. Ann Rech Vet 1978;9:309-318.

Cabello G, Levieux D, Lefairve J. The effect of intra-amniotic injections of thyroxine on the absorption of colostral IgG_1 by the newborn kid. Brit Vet J 1980;136:193-194.

Carbrey EA, et al. Recommended standard laboratory techniques for diagnosis of IBR, BVD, PI-3. Proc Ann Gen Mtg Am Assoc Vet Lab Diag, Oklahoma City, 1971.

Card CS, Spencer GR, Stauber EH, Frank FW, Hall RF, Ward ACS. The weak calf syndrome -- epidemiology, pathology and microorganisms removed. Proc 77th Ann Mtg US Anim Hlth Assoc 1974;77:67-72.

Carr DH. Chromosomal anomalies as a case of spontaneous abortion. Am J Obstet Gynecol 1967;97:283-293.

Chan WS, Daniels VG, Thomas AL. Premature cessation of macromolecule uptake by the young rat intestine. J Physiol 1973;231:112P-113P.

Cimprich RE. Differential diagnosis of neonatal diarrhea in domestic animals. Comp Cont Ed Pract Vet 1981;3:S26-S29.

Coid CR, Lansdown ABG, McFadyen IR. Fetal growth, retardation and low birthweight following infection in pregnancy. In: Coid CR, ed. Infections and Pregnancy. London: Academic Press, 1977;289-305.

Cope GE. Estrus synchronization and reproduction management of beef herds in the south. Proc 14th Ann Conv Am Ass Bov Pract 1982;No 14:104-107.

Dardillat J, Trillat G, Larvor P. Colostrum immunoglobulin concentration in cows: relationship with their calf mortality and with the colostrum quality of their female offspring. Ann Rech Vet 1978;9:375-384.

David JSE, Bishop MWH, Cembrowicz HJ. Reproductive expectancy and infertility in cattle. Vet Rec 1971; 89:181-185.

Davidson JN, Yancy SP, Campbell SG, Warner RG. Relationship between serum immunoglobulin values and incidence of respiratory disease in calves. JAVMA 1981;179:708-710.

Dennis SM. The effect of bacterial endotoxin on pregnancy. Vet Bull 1966;36:123-128.

Dennis SM. Laboratory diagnosis of infectious bovine abortion. JAVMA 1969;155:1913-1922.

Dennis SM. Perinatal lamb mortality in western Australia. 2. Non-infectious conditions. Aust Vet J 1974;50:450-452.

Dennis SM. Perinatal mortality of ruminants. Compend Cont Ed Pract Vet 1979;1:S17-S26.

Dennis SM. Investigating perinatal calf mortality. Proc Ann Mtg Soc Theriogenol. Omaha NE, September 10-12, 1980a, pp 150-168.

Dennis SM. Infectious bovine abortion in a practitioner's approach to diagnosis. Vet Med/SAC 1980b;73:459-466.

Dennis SM. Field methods for assisting the diagnosis of bovine abortion. Proc 11th Int Congr Dis Cattle. Israel, 1980c;498-503.

Dennis SM. Low viability of calves at birth. Vet Ann 1981;21:63-73.

Dennis SM. Perinatal pathology notes, Kansas State University, 1984;1-244.

Dierks RE, Smith MH, Gollehon D. Isolation and characterization of adenoviruses from aborted fetuses and calves with weak calf syndrome. Proc 19th Ann Mtg Am Assoc Vet Lab Diag 1976;395-404.

Doig PA, Ruhnke HL, Palmer NC. Experimental bovine genital ureaplasmosis. I. Granular vulvitis following vulvar inoculation. Can J Comp Med 1980;44:252-258.

Donald HP. Perinatal deaths among calves in a cross-bred dairy herd. Anim Prod 1963;5:87-95.

Done JT. Virus teratogens and domestic animals. Vet Ann 1978;18:1-12.

Done JT, Terlecki S, Richardson C, Harkness JW, Sands JJ, Patterson DSP, Sweasey D, Shaw IG, Winkler CE, Duffell SJ. Bovine virus diarrhea-mucosal disease virus - pathogenicity for the fetal calf following maternal infection. Vet Rec 1980;106:473-479.

Drost M. Perinatal care of the calf. In: Morrow DA, ed. Current Therapy in Theriogenology. Philadelphia: WB Saunders Co, 1980;274-276.

Drost M, Franco OJ, Shille VM, Thatcher MJ, Thatcher WW. The effect of pregnancy diagnosis per rectum on early embryonic death in the cow. Proc XII World Congr Dis Cattle 1982;1:623-627.

EEC. Calving problems and early viability of the calf. In: Hoffmann B, Mason IL, Schmidt J, ed. Commission of the European Communities, Coordination of Agricultural Research. Martinus Nijhoff Publishers, The Hague, 1979.

Ellis WA, Logan EF, O'Brien JJ. Serum immunoglobins in aborted and non-aborted bovine foetuses. Clin Exp Immunol 1978;33:136-141.

Ensminger ME, Galgan MW, Slocum WL. Problems and practices of American cattlemen. Wash Agr Exp Stat Bull 1955;562.

Evermann JF. Calfhood morbidity and mortality in the Northwestern United States 1977-1979. Proc 22nd Ann Mtg Am Assoc Vet Lab Diag 1979;379-394.

Francos G, Mayer E. Observations on some environmental factors connected with fertility in heat stressed cows. Theriogenology 1983;19:625-633.

Gay CC, McGuire TC, Parish SM. Seasonal variation in passive transfer of immunoglobulin G1 to newborn calves. JAVMA 1983;183:566-568.

Getty S, Ellis DJ. Experimental use of bull semen contaminated with Pseudomonas aeruginosa organisms. JAVMA 1967;150:1300.

Gopal T. Investigations into prenatal and perinatal mortalities among calves. PhD thesis, Kansas State University, 1977.

Grunsell GS. Mortality and morbidity in calves. Vet Rec 1956;68:788.

Hamilton GF, Turner AS, Ferguson JG, Pharr JW. Slipped capital femoral epiphysis in calves. JAVMA 1978; 172:1318-1322.

Haughey KG. Meningeal haemorrhage and congestion associated with the perinatal mortality of beef calves. Aust Vet J 1975;51:22-27.

Hawk HW. Beltsville Symposia in Agricultural Research. (3) Animal Reproduction. New York. Wiley J, ed. 1979;19.

Hillman RB. Bovine mycotic placentitis in New York state. Cornell Vet 1969;59:263-288.

Hindson JC. Quantification of obstetric traction. Vet Rec 1978;102:327-332.

Hoerlein AB, Jones DL. Bovine immunoglobulins following induced parturition. JAVMA 1977;170:325.

Hoerlein AB. Bovine genital vibriosis. In: Morrow DA, ed. Current Therapy in Theriogenology. Philadelphia: WB Saunders Co, 1980;479-482.

Horner GW, Johnson RH, Dennett DP, Lane WR. A serological study of bovine foetal immunoglobulins. Aust Vet J 1973;49:325-329.

House JA. Economic impact of rotavirus and other neonatal disease agents of animals. JAVMA 1978;173:573-576.

Hubbert WT, et al. Recommendations for standardizing bovine reproductive terms. Cornell Vet 1972;62:216-237.

Hubbert WT, Booth GD, Bolton WD, Dunne HW, McEntee K, Smith RE, Tourtellotte ME. Bovine abortions in five Northeastern states, 1960-1970; Evaluation of diagnostic laboratory data. Cornell Vet 1973;63:291-316.

Jerrett IV, McOrist S, Waddington J, Browning JW, Malecki JC, McCausland IP. Diagnostic studies of the fetus, placenta and maternal blood from 265 bovine abortions. Cornell Vet 1984;74:8-20.

Johnston NE, Oxender WD. Effect of altered serum glucocorticoid concentrations on the ability of the newborn calf to absorb colostral immunoglobulins. Am J Vet Res 1979;40:32.

Kahrs RF. Effects of infectious bovine rhinotracheitis on reproduction. In: Morrow DA, ed. Current Therapy in Theriogenology. Philadelphia: WB Saunders Co, 1980;492-497.

Kansas State Diagnostic Laboratory. Annual Report of Incidence of Infectious causes of Disease in Kansas. 1981.

Keeler RF. Alkaloid teratogens from lupinus, conium, veratrum and related genera. In: Effects of Poisonous Plants on Livestock. New York, NY: Academic Press, 1978;397-408.

Kendrick JW, McEntee K. The effect of artificial insemination with semen contaminated with IBR-IPV virus. Cornell Vet 1967;1:3-11.

Kendrick JW, Gillespie JH, McEntee K. Infectious pustular vulvovaginitis of cattle. Cornell Vet 1958;48:458-493.

Khera SS. Fetal and young calf mortality among bovine farmstock in India. Indian J Anim Sci 1981;51:292-302, 425-431, 432-438.

Kincaid CN. Survey of reproduction in S-10 breeding herds. Southern Region Technical Committee S-10, Improvement of Beef Cattle for the Southern Region through Breeding Methods, 1957.

Kirkbride CA, Bicknell EJ, Reed DE, Robl MG, Knudtson WU, Wohlgemuth K. A diagnostic survey of bovine abortion and stillbirth in the northern plains states. JAVMA 1973; 162:556-560.

Kirkbride CA. Abortive diseases of cattle: their significance and prevalence. Proc Ann Mtg Soc Theriogenol, St. Paul, MN, 1977;135-142.

Kirkbride CA. Abortive diseases of cattle: their significance and prevalence. Vet Med/SAC 1979;74:1151-1155.

Koger M, Mitchell JS, Kidder RW, Burns WC, Hentges JF, Warnick AC. Factors influencing survival in beef calves. J Anim Sci 1967;26:205.

Koch RM, Algeo JW. The beef cattle industry: changes and challenges. J Anim Sci 1983;57(Suppl. 2):28-43.

Kvasnicka W. Personal communication, Herd Health Veterinarian. US Meat Animal Research Center, Clay Center NE, 1984.

Larvor P. Treatment of the newborn calf. In: Hoffman B, et al, ed. Calving Problems and Early Viability of the Calf. Martinus Nijhoff Publ, The Hague, 1979;539-548.

Laster DB, Gregory KE. Factors influencing peri- and early postnatal calf mortality. J Anim Sci 1973; 37:1092-1097.

Laster DB, Glimp HA, Cundiff LV, Gregory KE. Factors affecting dystocia and the effects of dystocia on subsequent reproduction in beef cattle. J Anim Sci 1973;36:695.

Leipold HW. Genetics and disease in cattle. Proc 11th Ann Conv Am Assoc Bovine Pract, Baltimore, MD. December 1978;11-14, 18-31.

Leipold HW. Diagnosis and control of undesirable genetic diseases and lethal factors in cattle. Proc XI Intern Congr Dis Cattle, Spain. 1980;543-555.

Leipold HW, Dennis SM. Congenital defects affecting bovine reproduction. In: Morrow DA, ed. Current Therapy in Theriogenology. Philadelphia: WB Saunders Co, 1980;410-441.

Leipold HW, Huston K, Dennis SM. Bovine congenital defects. Adv Vet Sc Comp Med 1983;27:198-272.

Lindhe B. Dead and difficult births in cattle and measures for their prevention. World Rev Anim Prod 1966;4:53-58.

Logan EF, McMurray CH, O'Neill DG, McParland PJ, McRory FJ. Absorption of colosoral immunoglobulins by the neonatal calf. Brit Vet J 1978;134:258-262.

Lumba F, Fumiere I, Tshibangn M, Chauvaux G, Bienfet V. Immunoglobulin transfer to calves and health problems in large bovine units. Ann Rech Vet 1978;9:353-360.

Luedke AJ, Jochim MM, Jones RH. Bluetongue in cattle: effects of Culicoides variipennis transmitted bluetongue virus on pregnant heifers and their calves. Am J Vet Res 1977;38:1687-1695.

Luedke AJ, Walton TE. Effect of maternal breeding of heifers to a bluetongue virus carrier bull. In: Proc XI Int Congr Dis Cattle 1980;478-491.

Martin SW, Schwabe CW, Franti CE. Dairy calf mortality rate: characteristics of calf mortality rates in Tulane County, California. Am J Vet Res 1975a;36:1099-1104.

Martin SW, Schwabe CW, Franti CE. Dairy calf mortality rate: influence of meterologic factors on calf mortality rate in Tulane County, California. Am J Vet Res 1975b; 36:1105-1109.

McCormick WC, Southwell BL, Warwick EJ. Factors affecting performance in herds of purebred and grade Hereford cattle. Ga Agr Exp Sta Tech Bull 1956;N.S.5.

M'Fadyean J, Stockman S. Report Dept Comm Bd Agric Fish Part I, 1909.

McFarlane D. Perinatal lamb losses. 1. An autopsy method for the investigation of perinatal losses. NZ Vet J 1965;13:116-135.

McGuire TC, Pfeiffer NE, Weikel JM, Bartsch RC. Failure of colostral immunoglobulin transfer in calves dying from infectious disease. JAVMA 1976;169:713-718.

Mebus CA, Newman LE, Stair EL, Jr. Scanning electron, light and immunofluorescent microscopy of intestine of gnotobiotic calf infected with diarrhea coronavirus. Am J Vet Res 1977;36:1-19.

Miller RB. A discussion on reproductive failure in cattle. Bovine Pract 1982;17:39-51.

Miller RB, Quinn PJ. Observations on abortions in cattle: a comparison of pathological, microbiological and immunological findings in aborted foetuses and foetuses collected at abattoirs. Can J Comp Med 1975;39:270-290.

Miller and Graves, 1932. Cited by Roberts, 1971.

Miller RB, Lein DH, McEntee KE, Hall CE, Shin S. Haemophilus somnus infection of the reproductive tract of cattle: a review. JAVMA 1983;182:1390-1392.

Miller JM, VanDerMarten MJ. Reproductive tract lesions in heifers after inoculation with infectious bovine rhinotracheitis virus. Am J Vet Res 1984;45:790-794.

Mills KW, Tietze KL. Monoclonal antibody enzyme-linked immunosorbent assay for identification of K99-positive Escherichia coli isolates from calves. J Clin Microbiol 1984;19:498-501.

Moojen V, Roberts AW, Carter GR. Microbial causes of bovine abortion in Michigan. Vet Med/SAC 1983;78:102-106.

Moon HW. Mechanisms in the pathogenesis of diarrhea: a review. JAVMA 1978;172:443-448.

Muggli NE, Hohenboken WD, Cundiff LV, Kelley KW. Inheritance of maternal IgG_1 concentration by the bovine neonate. J Anim Sci 1983;57(Suppl. 1):162.

National Academy Science Subcommittee on Prenatal and Postnatal Mortality in Cattle, 1968; Publ no 1685, Washington, DC. pp 1-130.

NCA. The Future for Beef. A report of the Social Advisory Committee, National Cattleman's Association. Beef Business Bull, March 5, 1982, Englewood, CO.

Olson DP, Papasian CJ, Ritter RC. The effects of cold stress on neonatal calves. 1. Clinical condition and pathological lesions. Can J Comp Med 1980a;47:11-18.

Olson DP, Papasian CJ, Ritter RC. The effects of cold stress on neonatal calves. 2. Absorption of colostral immunoglobulins. Can J Comp Med 1980b; 44:19-23.

Olson DP, Bull RC, Kelley KW, Ritter RC, Woodard LF, Everson DD. Effects of maternal nutrition restriction and cold stress on young calves: clinical condition, behavioral reactions, and lesions. Am J Vet Res 1981;42:758-762.

Osburn BI, Stabenfeldt GH, Adams AA, Trees C, Sawyer M. Perinatal immunity in calves. JAVMA 1974;164:295-298.

Osburn BI, Muchuchlan NJ, Terrell TG. Ontogeny of the immune system. JAVMA 1982;181:1049-1052.

Oxender WD, Newman LE, Morrow DA. Factors influencing dairy calf mortality in Michigan. JAVMA 1973;162:458-460.

Oxender W, Adams W. Problems associated with calving and neonatal period in beef cattle. In: Mason et al., ed. Calving Problems and Early Viability of the Calf. Commission of the European Communities, Martin Nijhoff Publishers, The Hague, 1979;408-422.

Parsonson IM, Clark BL, Duffy JH. Early pathogenesis and pathology of Tritrichomonas foetus infection in virgin heifers. J Comp Path 1976;86:59.

Patterson DJ, Bellows RA, Burfening PJ, Short RE, Carr JB. Incidence and causes of neonatal and postnatal mortality in range cattle; USDA, SEA and Montana State University. J Anim Sci 1979;49(Suppl. 1):325.

Pfeiffer NE, McGuire TC. A sodium sulfite-precipitation test for assessment of colostral immunoglobulin transfer to calves. JAVMA 1977;170:809-811.

Philipsson J. Calving performance and calf mortality. Livest Prod Sci 1976;3:319-331.

Philipsson J, Foulley J, Lederer J, Liboriussen T, Osinga A. Sire evaluation standards and breeding strategies for limiting dystocia and stillbirth. Livest Prod Sci 1979;6:111-127.

Phillips R. Interpretation of serological results received from the Diagnostic Laboratory. Proc June Conference, College of Veterinary Medicine, Kansas State University, 1983, HI-4.

Plowright W, Kalunda M, Jessett DM, Herniman KAJ. Congenital infection of cattle with the herpesvirus causing malignant catarrhal fever. Res Vet Sci 1972;13:37-45.

Potgieter LND, McCracken MD, Hopkins FM, Walker RD. Effect of bovine viral diarrhea virus infection on the distribution of infectious bovine rhinotracheitis virus in calves. JAVMA 1984;185:1397.

Radostits DM, Acres SD. Disease of calves admitted to a large animal clinic in Saskatchewan. Can Vet J 1974;15:82.

Randall GCB. Perinatal mortality: some problems of adaptation at birth. Adv Vet Sci Comp Med 1978;22:53-81.

Rice LE. Perinatal management of calves. Proc Ann Mtg Soc Theriogenol, Mobile, Alabama, 1979;138-150.

Rice FJ, Woodward RR, Queensberry JR, Willson FS. Fertility of beef cattle raised under range conditions. Mont Exp Sta Bull 1961;561.

Rice LE, Wiltbank JN. Factors affecting dystocia in beef cattle. JAVMA 1972;161:1348-1358.

Richardson C. Intrauterine growth retardation in sheep. Vet Ann 1978;18:101-106.

Roberts SJ. Veterinary Obstetrics and Genital Diseases, 2nd ed., Publ by Author, Ithaca, NY, 1971;447-472.

Robinson JS. Growth of the fetus. Brit Med Bull 1979; 35:137-144.

Roche JF. Reproductive wastage following artificial insemination of heifers. Vet Rec 1981;109:401-404.

Roy JHB. The calf, 4th ed. Pub Butterworth Inc. 1980; 53-66, 369-370, 373-377.

Saed OM, Al-Aubaidi JM. Infertility in heifers caused by pathogenic strain of Mycoplasma bovigenitalium. Cornell Vet 1983;73:125-130.

Sawyer M, Osburn BI, Knight HD, Kendrick JW. A quantitative serologic assay for diagnosing congenital infections of cattle. Am J Vet Res 1973;34:1281-1284.

Schultz RD. Developmental aspects of the fetal bovine immune response: a review. Cornell Vet 1973;63:507-535.

Schultz RD, Dunne HW, Heist CW. Ontogeny of the bovine immune system. Infect Immunol 1973;7:981-991.

Scott GH. Immunoglobulin absorption in calf neonates with special considerations of stress. J Dairy Sci 1980; 63:681.

Speicher JA, Hepp RE. Factors associated with calf mortality in Michigan dairy herds. JAVMA 1973;162:463-466.

Stables JW. Genetic selection for case of calving. Bovine Pract 1979;14:102-107.

Stauber EH. Weak calf syndrome: a continuing enigma. JAVMA 1976;168:223-225.

Storz J, Young S. Bovine fetal infections with parvoviruses. Proc XI Int Cong Dis Cattle. Israel, 1980;474-477.

Storz J, Whiteman CE. Bovine chlamydial abortions. Bovine Pract 1981;16:71-75.

Stuart LD, Oehme FW. Environmental factors in bovine and porcine abortion. Vet Human Tox 1982;24:435-441.

Swift BL. Bovine parainfluenza-3 virus: experimental fetal disease. JAVMA 1973;163:861-862.

Tayler JC. Beef production in the EEC and the coordination of research by the commission of the European communities. Livest Prod Sci 1976;3:305-318.

Thornber PM. Ureaplasma association with bovine infertility in South-Western Scotland. Vet Rec 1982;111:591.

Thornberry H. Conclusions from the EEC Seminar on perinatal ill-health in calves. In: Mason et al., ed. Calving Problems and Early Viability of the Calf, Commission of the European Communities, Martin Nijhoff Publishers, The Hague. 1979;487-493.

Torres-Medina A. Effect of combined rotavirus and E. coli in neonatal gnotobiotic calves. Am J Vet Res 1984;45:643-651.

Tryphonas L, Hamilton GF, Rhodes CS. Perinatal femoral nerve degeneration and neurogenic atrophy of quadriceps femoris muscle in calves. JAVMA 1974;164:801-807.

Wiltbank JN, Warwick EJ, Vernon EH, Priode BM. Factors affecting net calf crop in beef cattle. J Anim Sci 1961; 20:409.

Woelffer EA. Diagnosis of bovine abortion. JAVMA December 1, 1972;161:1284-1287.

Woodward RR, Clark RT. A study of stillbirths in a herd of range cattle. J Anim Sci 1959;18:85.

Young JS. Breeding patterns in commercial beef herds. Aust Vet J 1968;44:350-356.

Young JS, Blair JM. Perinatal calf losses in a beef herd. Aust Vet J 1974;50:338-344.

Zaugg JL, Kuttler KL. Bovine anaplasmosis: in utero transmission and the immunologic significance of ingested colostral antibodies. Am J Vet Res 1984;45:440-443.

Zemjanis R. Repeat-breeding or conception failure in cattle. In: Morrow DA, ed. Current Therapy in Theriogenology. Philadelphia: WB Saunders Co, 1980;205-213.

TABLE 1 - Time of Death Classification Relative to Birth

1. Antepartum Deaths
   - APD 1...dead for a long time
   - APD 2...dead for > 12 hours
   - APD 3...dead for < 12 hours

2. Partum Deaths
   - Early in a birth of:
   - PD 1...moderate duration
   - PD 2...long duration
   - Middle in a birth of:
   - PD 3...moderate duration
   - PD 4...long duration
   - End of a birth of:
   - PD 5...moderate duration
   - PD 6...long duration
   - PD 7...short duration

3. Postpartum Deaths
   - Immediate PP deaths (IPPD)
   - PPD 1...did not breathe
   - PPD 2...breathed, did not walk — body fat not metabolized
   - PPD 3...walked, did not feed — body fat not metabolized
   - PPD 4...food not beyond small intestine — body fat not metabolized
   - Delayed PP deaths (DPPD)
   - PPD 5...breathed, did not walk — body fat partially metabolized
   - PPD 6...walked, did not feed — body fat partially metabolized
   - PPD 7...food not beyond small intestine — body fat partially metabolized
   - Late PP deaths (LPPD)
   - PPD 8...walked, did not feed — body fat fully metabolized
   - PPD 9...food not beyond small intestine — body fat fully metabolized
   - PPD 10..body fat not metabolized — food has passed through whole of G.I. tract
   - PPD 11..body fat partially metabolized — food has passed through whole of G.I. tract
   - PPD 12..body fat fully metabolized — food has passed through whole of G.I. tract

From McFarlane (1965), modified by Dennis (1981)

TABLE 2 - Diagnosis of infectious causes of bovine abortion by percent and geographic area of U.S.

	Northeast	Northern Plains		Kansas	Michigan
	Hubbert et al. 1973	Kirkbride et al. 1973	Kirkbride 1979	KSU Dx. Lab 1981	Moojen et al. 1983
Mycotic	3.2	3.5	8.4	0.4	1.0
IBR virus	2.9	16.0	8.8	2.2	8.2
BVD virus	0.5	0.5	0.1	3.8	3.1
PI-3 virus	0.1	-	-	0.4	4.1
Coryne-bacteriosis	2.3	2.7	4.4	1.0	6.1
Leptospirosis	2.5	0.6	1.2	12.9	7.1
Campylo-bacteriosis	2.5	3.0	1.6	0.4	5.1
Strepto-coccosis	2.8	-	0.2	-	-
Total diagnosed	23.3	35.3	34.0	24.4	43.8

TABLE 3 - Gestational age and fetal characteristics*

Days of gestation	Body weight	Crown-Rump Length (cm)	External Appearance
60	8-30g (.25-1oz)	6-8 (2.5-3.25")	Claw buds and small scrotum recognizable, eyelids cover eyes
90	200-400g (6-13oz)	13-17 (5-6.5")	Hooves firm, hair on lips, chin and eyelids, scrotum present
120	0.8-2.0kg (2-4 lbs)	22-32 (8.5-12.5")	Hooves opaque, horn pits appear, fine hair on eyebrows
150	3.4kg (6.5-10lbs)	30-45 (12-17.5")	Hair on eyebrows and lips, testes in scrotum, teats well formed
180	5-10kg (11-12lbs)	40-60 (15.5-24")	Hair on inside of ear, around horn pits, tip of tail and muzzle
210	8-18kg (17.5-40lbs)	60-75 (24-30")	Hair on metatarsal/carpal and phalangeal regions, on back, long hair on tip of tail
240	15-25kg (33-55lbs)	65-85 (26-34")	Fine short hair all over Incisor teeth not erupted
270	20-50kg (44-110lbs)	80-100 (32-40")	Hair coat complete and long Incisor teeth erupted

*From Roberts, 1971 and Hubbert et al., 1972

Fig 1 - Summary of the various factors affecting PCM and the sequelae modified from Dennis (1980a, 1981).

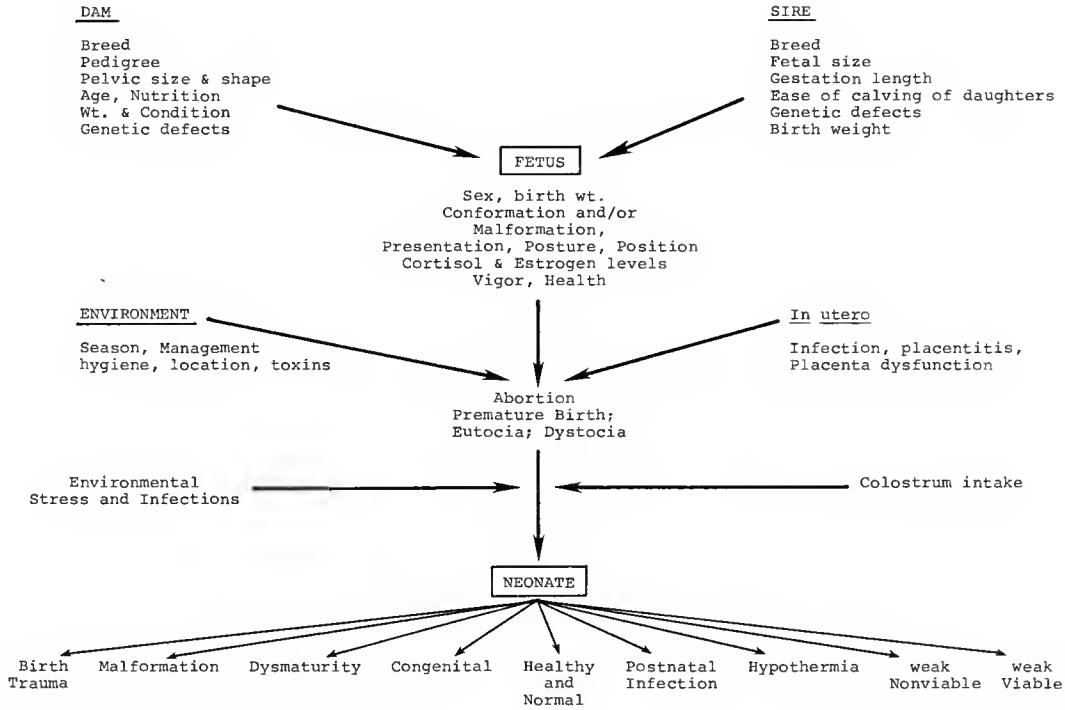

DAM
Breed
Pedigree
Pelvic size & shape
Age, Nutrition
Wt. & Condition
Genetic defects
SIRE
Breed
Fetal size
Gestation length
Ease of calving of daughters
Genetic defects
Birth weight
FETUS
Sex, birth wt.
Conformation and/or
Malformation,
Presentation, Posture, Position
Cortisol & Estrogen levels
Vigor, Health
ENVIRONMENT
Season, Management
hygiene, location, toxins
In utero
Infection, placentitis,
Placenta dysfunction
Abortion
Premature Birth;
Eutocia; Dystocia
Environmental
Stress and Infections
Colostrum intake
NEONATE
Birth Trauma
Malformation
Dysmaturity
Congenital
Healthy and Normal
Postnatal Infection
Hypothermia
weak Nonviable
weak Viable

Fig 2 - Summary of non-infectious causes of bovine abortion.

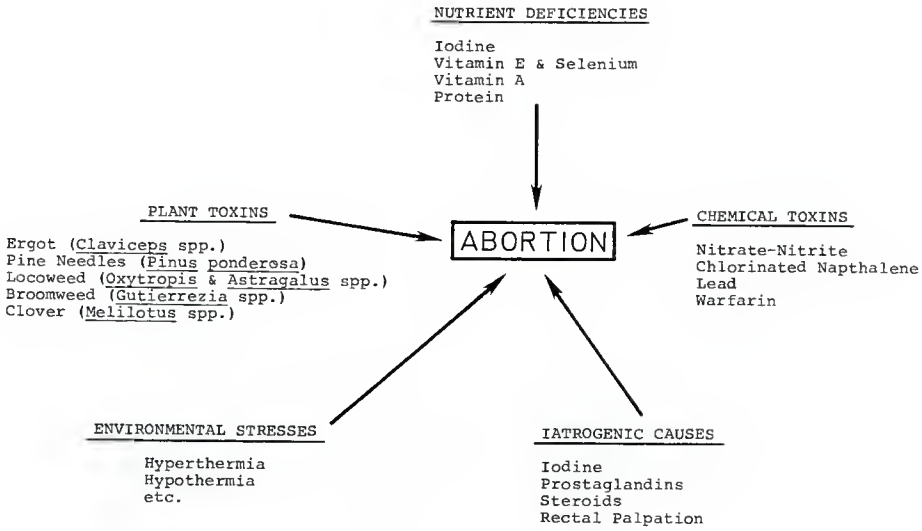

NUTRIENT DEFICIENCIES
Iodine
Vitamin E & Selenium
Vitamin A
Protein
PLANT TOXINS
Ergot (Claviceps spp.)
Pine Needles (Pinus ponderosa)
Locoweed (Oxytropis & Astragalus spp.)
Broomweed (Gutierrezia spp.)
Clover (Melilotus spp.)
ABORTION
CHEMICAL TOXINS
Nitrate-Nitrite
Chlorinated Napthalene
Lead
Warfarin
ENVIRONMENTAL STRESSES
Hyperthermia
Hypothermia
etc.
IATROGENIC CAUSES
Iodine
Prostaglandins
Steroids
Rectal Palpation

Fig 3 - Summary of infectious bovine abortion and specific and non-specific placental and fetal lesions. modified from Dennis (1980a).

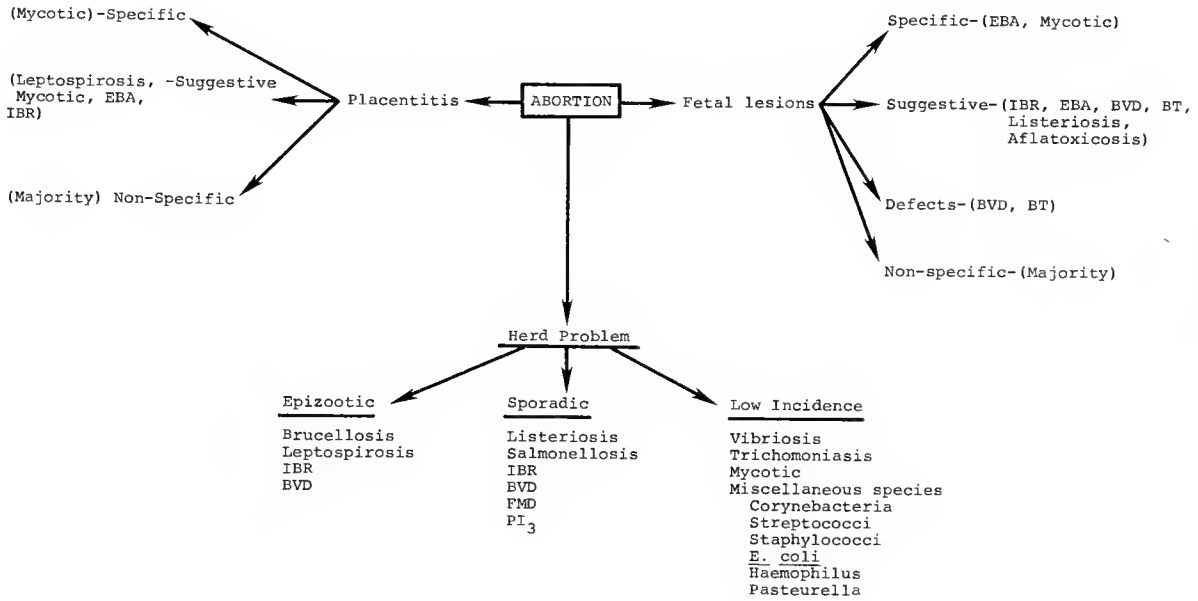

(Mycotic)-Specific
(Leptospirosis, -Suggestive
Mycotic, EBA,
IBR)
(Majority) Non-Specific
Placentitis
ABORTION
Fetal lesions
Specific-(EBA, Mycotic)
Suggestive-(IBR, EBA, BVD, BT,
Listeriosis,
Aflatoxicosis)
Defects-(BVD, BT)
Non-specific-(Majority)
Herd Problem
Epizootic
Brucellosis
Leptospirosis
IBR
BVD
Sporadic
Listeriosis
Salmonellosis
IBR
BVD
FMD
PI3
Low Incidence
Vibriosis
Trichomoniasis
Mycotic
Miscellaneous species
Corynebacteria
Streptococci
Staphylococci
E. coli
Haemophilus
Pasteurella

Fig 4 - Summary of diagnosis of intrauterine growth retardation or dysmaturity.

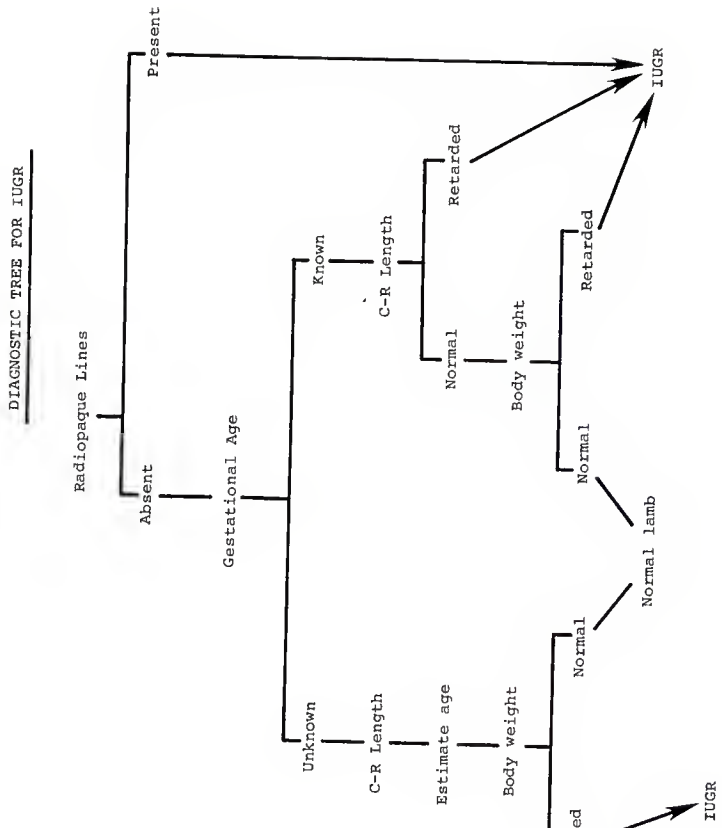
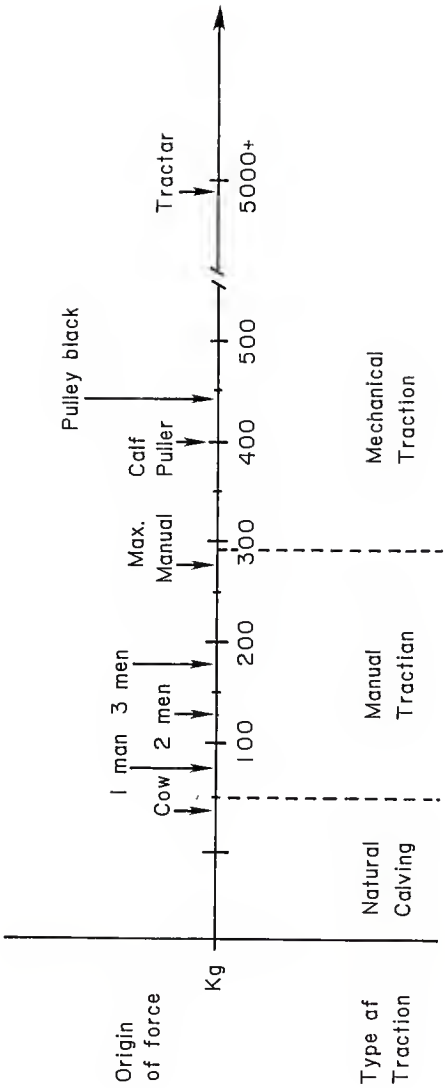

DIAGNOSTIC TREE FOR IUGR
Radiopaque Lines
Present
IUGR
Absent
Gestational Age
Known
C-R Length
Retarded
Normal
Body weight
Retarded
Normal
Unknown
C-R Length
Estimate age
Body weight
Normal
Normal lamb
Retarded
IUGR

Fig 5 - Amount of force (kg) exerted on the fetus by different types of traction.


Origin of force
Kg
Type of Traction
Natural Calving
Manual Traction
Mechanical Traction
Cow
1 man
2 men
3 men
Max. Manual
Calf Puller
Pulley black
Tractor
100
200
300
400
500
5000+

II. PERINATAL CALF MORTALITY IN THE KANSAS FLINT HILLS: INVESTIGATING INCIDENCE, DISTRIBUTION AND GENERAL CAUSES

INTRODUCTION

Perinatal calf mortality (PCM) is a complex problem resulting from a variety of causes and their interactions including management, climate, nutrition, behavior, infections, toxins, congenital defects, predation, genetics and unknown factors. Studies and surveys over the past 50 years reported the incidence of PCM in beef herds to range from 3.5 to 25.0% with an average of 8.6% of all females diagnosed as being pregnant (Baker and Quesenberry, 1944; Ensminger et al., 1955; Grunsell, 1956; McCormick et al., 1956; Kincaid, 1957; Woodward and Clark, 1959; Rice et al., 1961; Wiltbank et al., 1961; Kroger et al., 1967; Nat. Acad. Sci., 1968; Young, 1968; Rice and Wiltbank, 1972; Laster et al., 1973; Laster and Gregory, 1973; Young and Blair, 1974; Philipsson, 1976; Tayler, 1976; Dennis, 1979, 1980; E.E.C., 1979; Patterson et al., 1979; Thornberry, 1979; Roy, 1980; Khera, 1981; Roche, 1981; Cope, 1982; Miller, 1982). During this half century, the incidence of PCM has not appreciably improved. Beef cattle production has been intensified but in light of advances in technology, nutrition, antibiotics, husbandry, infectious disease diagnosis and control, preventive medicine, and therapeutic agents this lack of significant improvement is disappointing.

There has been a significant reduction in human perinatal mortality because the problems encountered by human neonates have been accurately defined and effective preventive measures developed (Randall, 1978). It is unlikely that PCM will be significantly reduced until the problems are better defined (Randall, 1978). Preventive measures to reduce PCM will result in only moderate and unpredictable success until the actual problems and causes within geographical areas are accurately identified.

Recently new insights into the relative importance of factors influencing PCM have been provided by necropsy findings correlated with fetal, natal and neonatal problems and their time-of-death (Dennis, 1979). There may be geographical differences in the distribution of PCM based on time-of-death (Young and Blair, 1974; Patterson et al., 1979).

PCM is influenced by events occurring during three critical periods: fetal, natal and neonatal (Dennis, 1980). Young and Blair (1974) reported the corresponding percent of PCM within these periods from their study to be 3.7, 60.5 and 35.8 percent, respectively.

This study was undertaken to investigate PCM in the Flint Hill area of Kansas over a three year period from 1982 to 1984. The main objective was to determine, define and clarify the incidence, distribution, and general causes of PCM in beef herds by systematic necropsy and time-of-death classification.

MATERIALS AND METHODS

Definitions

A lack of standardized definitions for evaluating PCM makes comparison between various studies difficult. Where applicable, the terms used in this study were recommended by the Committee on Bovine Nomenclature (Hubbert et al., 1972).

Perinatal Mortality: Death of fetuses and newborn calves after the embryonic period (approximately the first 45 days of gestation), during birth or within the first four weeks of life.

Fetal Death: Death of a fetus prior to the complete expulsion or extraction from the dam. Fetal deaths are subdivided into antepartum and parturient (natal).

Natal Nonviability: Fetus dying early in parturition with no signs of viability.

Neonatal Death: Death of a newborn occurring after birth and within the first four weeks of life.

Incidence: Number of calves lost in a herd during the perinatal period divided by the number of females diagnosed pregnant expressed as a percentage.

Distribution: Percent of PCM incidence distributed into the time-of-death categories: antepartum, partum, and postpartum.

General Cause: Classification of each death into one of three general causes: infectious, non-infectious, or undetermined.

Condition: Problem title for a problem-oriented approach to diagnosis.

Calf Populations

Data from necropsies were accumulated from two large general populations of calf deaths within the Flint Hills geographic region.

1. Calf deaths from area ranches agreeing to submit all PCM's for necropsy examination (contract calves).

2. Calf deaths submitted for necropsy from other herds (non-contract calves).

Ten beef cattle herds, five commercial and five pure-bred, agreed to submit all dead calves for examination. Each year that a ranch participated was considered as one ranch year. Seven of the 10 herds were privately owned and managed ranches and the remaining three were owned by Kansas State University (Department of Animal Science and Industry), but managed separately. All herds were separately located within the geographic and vegetative region known as the Kansas Flint Hills. All herds were within an 80 mile radius of Manhattan Kansas (Appendix 1).

Herd production and management information was also acquired by sending out a detailed survey (Appendix 2). Only spring calving cows and heifers were included with the calves being born from January thru June, depending on the husbandry practiced, the majority being born in March and April. Cattle breeds were primarily Hereford, Angus, Simmental, and their crosses for cows and Hereford, Angus, Simmental and Longhorn for bulls. The breeding season varied from 45 to 120 days and both natural service and artificial insemination were used.

Husbandry practices included cows and calves being grazed on native blue stem pastures from May to November, summer stocking rates of 6 to 7 acres per cow-calf, calf weaning in the fall, cows being moved to harvested fields to utilize crop residues until calving, cows and heifers being supplemented during winter with various types of roughage, protein cakes, energy cakes, grain, salt, and minerals, and cows being moved to protected pastures, fields or wheat pastures, for calving. Some ranches maintained cows on pasture the whole time. The average cow unit requirement per year in the Flint Hills is 8 to 10 acres.

Necropsy

A standard necropsy request form giving history and general information was completed for all calves submitted for necropsy (Appendix 3). Information usually included age or parity of dam, and dam and calf identification numbers. A necropsy number was assigned and the calf weighed to the nearest pound. Weight of calves delivered by fetotomy or with evidence of predation were not recorded.

A standardized necropsy procedure was followed, and all findings recorded on a special perinatal calf necropsy examination form (Appendix 4). A summary of necropsy findings and tentative diagnosis were then recorded on the necropsy form in triplicate and copies sent to the consulting veterinarian and the diagnostic laboratory.

Time-of-Classification

Time-of-death (TOD) classification was determined for all calves submitted for examination according to the criteria described by McFarlane (1965) (Appendix 5).

General Cause and Disease Condition

Classification as to three general causes or nature of death was made on all calves: infectious, non-infectious or undetermined. Infectious cause was determinated on the basis of gross and microscopic findings and/or diagnostic tests indicating an infectious agent or agents as the cause of death. Non-infectious cause was determinated on history and gross and microscopic findings suggestive of non-infectious agents (environmental, toxic, genetic, etc.) as the cause of death. Undetermined cause was made by lack of significant gross or microscopic changes or diagnostic test findings indicating an infectious or non-infectious cause.

Disease condition determination was made on all calves necropsied as the first of a three part problem-oriented approach to diagnosis: disease condition, gross diagnosis(es), and specific cause(s). Disease condition was divided into 10 general problems: abortion, acute death, birth trauma, neurologic dysfunction, disease complicated starvation, congenital defects, dystocia, natal non-viability, neonatal weakness, and uncomplicated starvation. History and gross findings were used for determining the diagnostic condition for each calf. Collection and handling

of tissues and specimens from the necropsied calves are summarized in Appendix 6.

In calves where the problem could be defined more specifically one or both of the two remaining parts were defined, for example, disease complicated starvation/ enteritis/rotavirus.

Data Analysis

All data collected for each calf was summarized on a data entry sheet (Appendix 7) and inputed into a Zenith 150 PC mircocomputer[a] using the file program PC-File III[b]. After the data was accumulated and edited it was sent via modem to a mainframe computer for statistical analysis using the Statistical Analysis Systems (S.A.S.) program to carry out comparisons and computations.

Statistical techniques used for data analysis were contingency tables, frequencies, chi-square and analysis of variance.

Contract Herd Reports

At the end of each calving season, a summarized report of the necropsies performed was sent to each herd participating in the PCM project (Appendix 8).

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[a]Zenith 150 PC microcomputer, Zenith Data Systems Corporation, St. Joseph, MI 49085

[b]PC-File III, Jim Button, ButtonWare, P.O. Box 5786, Bellevue, WA 98006

RESULTS

A total of 237 calves were examined by necropsy during the three year study; 155 contract and 82 non-contract. Classification by time-of-death is given in Table 1. Analysis of the TOD findings revealed five distinct categories: antepartum, early partum, late partum, early postpartum, and late postpartum (Table 2). Further analysis of the findings were confined to these five functional categories; the gross criteria are summarized in Appendix 9.

Incidence of Perinatal Calf Mortality

A total of 294 dead calves from 3,934 heifers and cows expected to calve in the contract herds were reported and 155 of the calves were necropsied (52.7%). During the first year of the study, 9 ranches participated, 9 in the second year, and 8 in the third, a total of 26 ranch years.

The mean incidence of PCM for each of the three years and the averages are depicted in Fig 1. The average PCM incidence calculated from the means for all ranch years was 7.99%.

Actual yearly incidence of PCM approached significance ($P \leq .06$) with 1982 and 1983 being significantly different ($P < .05$). However, the effect of year alone on incidence could not be determined as PCM incidence was influenced by size of cow herd and type of operation.

There was a difference between actual incidence of PCM over the three year period between purebred and commercial operations, 6.88% vs. 9.43%, respectively. This difference was not significant due to the variance within the groups and the influences of cow herd size. When adjusted for size of the cow herd, the average PCM incidence for commercial vs. purebred operations changed to 11.30% and 7.26%, respectively. This was significantly different at the $P < .06$ level (Fig 2).

Cow herd size over the three years ranged from 16 to 437. The average commercial operation tended to be larger than the average purebred operation, 212 vs. 100 cows per year, respectively. When the incidence of PCM for the type of operation was examined with respect to cow herd size there was a significant difference ($P < .05$). In purebred operations PCM incidence was not significantly effected by size of herd, where as, in commercial operations there was a highly significant negative correlation ($P < .001$) between incidence of PCM and the herd size (Fig 3).

Distribution of Perinatal Calf Mortality within Contract Herds

Environmental conditions, predatation, severe post-mortem autolysis and owner workload prevented almost half of the contract dead calves from being examined (47.3%). Analysis of distribution of PCM from herds where a high percent of the dead calves were examined compared to that from herds where only a low to moderate percent of the deaths were

examined revealed no significant difference. Therefore, all calves from contract herds were used to assess the area distribution of PCM.

The distribution of PCM by TOD for each year and the three year average is illustrated in Fig 4. For years 1983 and 1984, there was a significant difference in the distribution of PCM ($P \leq .01$). This difference was mostly due to yearly changes in the antepartum, early partum and late partum deaths compared to the remaining two death categories. The largest differences within TOD categories for 1983 and 1984 were antepartum (5.8% vs. 11.4%), early partum (3.5% vs. 15.9%) and late postpartum (40.2% vs. 15.9%).

When antepartum, partum and postpartum categories were compared for yearly influences, there were highly significant differences between all three years ($P < .01$). In 1982, 50% of deaths occurred during the early postpartum period, in 1983, 40.2% during the late postpartum period, and in 1984, 45.5% were parturient.

In comparing PCM distribution to the type of operation, commercial or purebred, there was no significant difference. However, cow herd size had a very significant effect on the average distribution of PCM ($P < .0005$) (Fig 5). The largest variances between small (< 150 cows) and large (> 150 cows) herds occurred within the antepartum and late partum categories (8.2% vs. 4.3% and 42.4% vs. 11.4%,

respectively). The majority of losses in smaller herds occurred during late partum in contrast to late postpartum in larger herds.

The average distribution of PCM was compared to the general causes of death (infectious, non-infectious, and undetermined) to assess trends or differences. Accurate analysis was not possible using all time-of-death classes due to the lack of numbers, however, there appeared to be a significant trend. Comparison of antepartum, partum and two postpartum categories to general causes could accurately be analyzed and proved to be significantly different ($P \leq .0001$) (Table 3).

The cause of most antepartum deaths were undetermined. Partum deaths were primarily due to non-infectious causes (91.1%). Non-infectious causes were found at the highest frequency (55.4%) during the natal period. Most early postpartum deaths were non-infectious but this category included the highest frequency of undetermined causes (56.0%). Deaths during the late postpartum period were primarily infectious (63.8%). This category also contained the highest frequency of infectious causes (78.9%).

The most frequent condition or problem associated with each of the five TOD categories was examined and the frequency of the general cause was noted (Table 4 and 5). In the antepartum category, all deaths were associated with abortion; 30% of the abortions were infectious, 10.0% non-infectious, and 60.0% unknown.

Natal nonviability (83.3%) was the major condition found in the early partum category. The corresponding general cause breakdown was 20.0% infectious, 60.0% non-infectious, and 20.0% undetermined. In the late partum category, dystocia and birth trauma accounted for 97.7% of the deaths.

Early postpartum deaths resulted primarily from neonatal weakness (71.4%) or birth trauma (16.7%). The general causes of neonatal weakness were found to be 8.8% infectious, 55.9% non-infectious, and 35.3% undetermined. The late postpartum category had the most diverse etiology: disease complicated starvation accounted for 55.3% of the deaths, uncomplicated starvation for 14.9%, and acute deaths 12.8%. Eighty-eight percent of disease complicated starvation conditions were caused by infectious agents and 11.5% from non-infectious causes. All uncomplicated starvation deaths were due to non-infectious causes. Of the acute deaths, 42.6% resulted from infectious agents, 42.6% from non-infectious causes, and 14.3% undetermined.

Distribution of Perinatal Calf Mortality in Non-Contract Calves

Time-of-death distribution of 82 calves necropsied from non-contract herds was determined to see if random submission was representative of the actual PCM distribution occurring in the region. The difference was very highly significant ($P \leq .0001$) (Fig 6). Deaths examined from non-contract herds mainly involved the antepartum and late

postpartum periods (77%). They varied significantly from each other and from the remaining three categories ($P \leq .0001$).

Within the non-contract population, there was no significant annual influence on PCM distribution. Comparison of distribution by general cause could not accurately be determined because of the relatively small number of calves necropsied. The frequency of general cause within antepartum and late postpartum categories was: antepartum deaths (65.0% infectious agents, 5.0% non-infectious, and 30.0% undetermined) and late postpartum deaths (76.7% infectious, 13.9% non-infectious, and 9.3% undetermined).

The conditions most common in each TOD category were similar to those found in the contract calf population (Table 2). The few exceptions to the general pattern were not significant due to insufficient numbers. However, for most conditions identified within the non-contract calf population, infectious causes were the most frequent.

General Causes of Perinatal Calf Mortality

General causes of death over the three year period were compared between contract and non-contract calf populations and the difference was highly significant ($P \leq .0001$) (Fig 7). The extreme variation between these two populations came from differences in the frequency of calves dying from non-infectious and infectious causes. Contract calf causes of death were non-infectious 59.4% compared to 19.5%

for non-contract calves. Conversely, non-contract general cause of death was infectious 59.8% compared to 24.5% for contract calves. Since there was such an extreme variation between populations, further analyses were done separately.

General causes of death for contract calves were not significantly influenced by year or type of operation. However, the relationship between cow herd size and general cause of death approached significance ($P \leq .09$). Non-infectious causes of PCM were most prevalent in both small (≤ 150) and large (> 150) cow herds. Smaller operations tended to have a higher incidence, 64.7% vs. 52.9%. Infectious causes of calf mortality tended to be a problem more in herds over 150 cows, 32.9% vs. 17.7%. Undetermined causes of death tended to be higher in herds with less than 150 cows, 17.7% vs. 14.3%, and within this herd size, the frequency equated with infectious causes.

The most frequent infectious cause within the contract calf population was complicated starvation (60.5%) mostly due to enteritis or septicemia. Within the non-infectious category, dystocia (42.4%) and neonatal weakness (20.7%) were the two major conditions related to PCM. Neonatal weakness of non-infectious was generally due to exposure. Of the undetermined causes of perinatal death, neonatal weakness was the major condition (48%) followed by abortions (24%). The major condition associated with PCM without regard to general cause or TOD was dystocia (25.8%), followed by neonatal weakness (21.9%), and then disease complicated starvation (16.8%).

Within the non-contract population of calves examined, complicated starvation, abortion and acute deaths were the major infectious conditions encountered; 34.7%, 26.5% and 24.5%, respectively.

Similarities were present in the major conditions associated with general cause within the two calf populations but there was a large discrepancy in frequency (Tables 6 and 7). The major condition randomly selected from necropsy was either abortion (24.4%) or disease complicated starvation (24.4%), followed by neonatal weakness (18.3%), and acute death (15.9%). Even though there were extreme frequency variances for non-infectious causes of death between the two calf populations, dystocia was the major non-infectious condition for both, contract (25.8%) and non-contract (37.5%). The two calf populations were similar regarding the major conditions of undetermined cause: neonatal weakness (contract 48% and non-contract 47.1%) and abortions (contract 24% and non-contract 35.3%).

DISCUSSION

The diagnostic approach of the study was to systematically necropsy and classify all dead calves by their time-of-death relative to birth (McFarlane, 1965) and to use the results as a basis for comparing PCM in individual herds. TOD was used to identify and define PCM problems and to monitor their changes. Initially, calves were classified by one of the 22 different TOD classes (Dennis, 1980) but on

analysis, this was modified to five functional categories: antepartum, early partum, late partum, early postpartum, and late postpartum. These categories along with codes and diagnoses were utilized for computer analysis, comparisons, and computations.

The average incidence of PCM over the three year period of 7.99% was slightly lower than the reported average of approximately 8.6% in the literature. A mortality rate of 5% seems to be economically acceptable (Oxender and Adams, 1979; Roy, 1980). We are still far above this goal. Rates of less than 1% are technically feasible and similar to infant mortality (Randall, 1978), but they will not be achieved unless they are economically beneficial to producers.

The incidence derived from this study may be a more accurate estimate of PCM of beef cattle than other more recent studies conducted on government or state owned and managed herds (Laster and Gregory, 1973; Young and Blair, 1974; Patterson et al., 1979). Conditions and trends in these herds are not always indicative of problems in privately owned herds. Factors such as economics, labor resources, nutrition, breeding strategy, and facilities influence management and productivity and these factors may vary significatly between government or state owned and privately owned operations. Young and Blair (1974) noted a two-fold increase in calf mortality among multiparous cows compared to previous work they had done using privately owned commercial herds (Young, 1968).

There was significant variation in PCM incidence between the three years. If the study would have been carried out over a longer period of time, the differences between the reported incidence and the Flint Hills area incidence may not have been so large; it may have been larger. The weather patterns, temperature and moisture during the calving seasons over the three years were variable. Further research into the effects of different meterological variables on beef PCM is needed. General meterological factors effecting dairy calf mortality in the U.S. (Martin et al., 1975b), and dairy, Zebu and buffalo PCM in India (Khera, 1981) have been studied.

Of interest was the effect of size of cow herds within commercial and purebred operations on PCM incidence. In purebred operations PCM incidence did not vary significantly with increasing cow herd size. In commercial operations, however, as cow herd size increased, PCM incidence significantly decreased. Both observations are true only within the limits of the data collected (16 cows herd minimum and 437 cows herd maximum). When the data was adjusted to remove the influence of cow herd size, there was a significant trend for purebred operations to have a lower overall PCM incidence when compared to commercial operations. Many reasons can be postulated for these observations, the major one being that the value of an individual purebred calf may be up to ten times greater.

The effect of labor and management personnel significantly influence dairy PCM (Speicher and Hepp, 1973; Martin et al., 1975a). This is most likely the case in beef cattle PCM also; however, further research is needed to be done to determine the actual effect. The general statement that PCM in dairy cattle tends to increase as herd size increases did not hold true in this beef cattle study (Speicher and Hepp, 1973).

The distribution of and general causes of PCM differed significantly between calf populations indicating that results derived from calves randomly selected at necropsy are not indicative of the true position in the general population. Data from the non-contract calf population was biased towards infectious and undetermined causes of death and antepartum or late postpartum times of death. For indepth studies of these problems a population like this serves as an excellent model. However, if trying to assess general information, distribution and causes of PCM for an area a number of contract herds are necessary. It is best if a high percentage of all calf deaths in the contract herds are examined. In this study, there was no significant difference in distribution of PCM in contract herds where a low or moderate percent of the deaths were examined as compared to herds where a high percent of the deaths were examined. Ideally, all dead calves in a herd should be necropsied.

The area distribution of PCM into the critical fetal, natal and neonatal periods were 6.5%, 36.1% and 57.4%, respectively. This varied significantly from reported distribution in the Australian beef cattle study of 3.7%, 60.5%, and 35.8% (Young and Blair, 1974). The distribution of PCM in the neonatal period was similar to the 62% reported by Patterson et al. (1979). Comparative analysis using the five TOD classes proved to be rewarding. Distribution by TOD was significantly influenced by year (1983 and 1984), cow herd size, and general cause. Type of operation did not significantly influence TOD distribution.

Antepartum deaths comprised 6.5% of the total PCM incidence. Essentially all were due to abortion and there was considerable variance from year to year. Smaller herds experienced almost twice as many abortions than the larger herds. This was probably due to the smaller areas that the cows were kept and the closer cow to cow contact more of the time. Purebred operations had a four fold increase in antepartum deaths over commercial operations. This may have resulted from more frequent introduction of new animals and transportation of existing animals in purebred operations, and some antepartum deaths being unobserved in some commercial units due to extensive management and the lower value of the calves.

Forty percent of the abortions were diagnosed: 30% infectious, 10% non-infectious, and 60% undetermined. Diagnostic efficiency was better in the non-contract calf

population where a higher percent were determined to be infectious and only 30% were undetermined. This suggested that, when extensive diagnostic laboratory tests were used, a fairly high percent of infectious abortions can be identified. The percent undetermined may be due to non-infectious causes that are poorly understood or unknown factors that have not been determined. In light of the substantial variance from year to year, the former seems more reasonable. Further research into these areas is warranted.

Partum deaths comprised approximately one-third of the total PCM incidence; early partum (7.7%) or late partum (28.4%). The incidence was substantially less than that of some earlier reports (Laster and Gregory, 1973). Most of the decrease apparently resulted from management changes in breeding and labor to decrease the incidence of dystocia. By using TOD, it was possible to identify early partum deaths as a problem; the incidence was larger than that for antepartum deaths.

Early partum deaths, like antepartum deaths, varied substantially from year to year and by size of cow herd operation. Most deaths were due to natal nonviability and both infectious and undetermined causes accounted for 20% each. The incidence of infectious natal nonviability were similar to that of abortion. Many calves could have been sublethally infected or injured and survived instead of aborting, only to lack the viability to withstand the initial stages of parturition (Haughey, 1975).

Late partum deaths continue to be a major cause of PCM. The deaths can be decreased but will require management changes to reduce the incidence of dystocia and birth trauma. There have been many suggestions on how to reduce PCM due to dystocia and birth trauma (Laster and Gregory, 1973; Bar-Anan, 1979; Berg, 1979). How much reduction can be made will vary as economic thresholds on most operations dictate whether it is profitable to reduce the incidence any further. This should be determined individually and then set as a goal for that herd. The only substantial variance in the frequency of late partum deaths was found on comparing it to the size of operation. Smaller operations had almost a four fold increase in deaths from dystocia and birth trauma compared to larger operations. The reasons for this were that smaller herds tended to calve out a higher percentage of heifers than larger herds, and larger breed sires (eg. Simmental) were used more frequently in the smaller herds than the larger ones. Cow herd and calf management personnel probably also had an influence as larger herds generally utilize a more frequent cow checking procedure during calving with more than one person. The effect of cow-calf management personnel could not be determined in this study.

The greatest PCM losses occurred during the postpartum period, with early postpartum deaths and late postpartum deaths accounting for nearly equal proportions (27.1% vs. 30.3%). By comparing the TOD with the general condition, it

was found that neonatal weakness accounted for nearly three-fourths of the losses within the early postpartum category. A variety of general conditions made up the late postpartum category. Neonatal weakness primarily resulted from non-infectious factors but a substantial proportion were undetermined. Of the non-contract calf population, neonatal weakness of undetermined cause was most frequent and infections next. Neonatal weakness also was responsible for approximately 10% of the deaths in both calf populations in the late postpartum class.

There was substantial variation in annual frequency of early and late postpartum deaths. This finding emphasized the importance of environmental conditions on neonatal calf survival. Even though the three year average revealed no substantial difference between frequency of these two post-parturient classes, there appeared to be a negative annual correlation or relationship. Their frequency within the postpartum period was approximately one third vs. two thirds with the frequency alternating between the two classes yearly. Neither class of postparturient losses were influenced by size or type of operation. Infectious causes of PCM were most prevalant during the late postpartum period. Over 60% of the infectious deaths were complicated by starvation with enteritis being the most common diagnosis.

The largest single cause of PCM was dystocia, a finding that confirmed previous reports (Young and Blair, 1974; Patterson et al., 1979). Dystocia was also the major non-infectious condition identified in this study.

Neonatal weakness was the second largest cause of PCM. The overall incidence of neonatal weakness was nearly as high as that of dystocia. The incidence was significantly higher than that reported by Khera (1981). Neonatal weakness was also the major undetermined cause. The results indicated that neonatal weakness is a major problem in PCM. Due to the high incidence of undetermined causes associated with neonatal weakness, further research is needed to clarify the problem.

Neonatal weakness is similar to the condition reported in beef calves as weak calf syndrome (WCS). The reported gross findings of WCS (Stauber, 1976), were similar to those associated with neonatal weakness from exposure. This was reported previously (Olson et al., 1981).

Disease complicated starvation was the third major cause of PCM. It was also the major infectious condition identified in this region. Most cases of disease complicated starvation were due to enteritis or septicemia. Reasons for the high incidence of infectious disease during the late postpartum period and its resultant effect on the nutrition of the calf are many. Over 50% of the newborn beef calf problems are related to the digestive system (Radositis and Acres, 1974). Laster and Gregory (1973) found that dystocic calves have a four times greater chance of dying later than those not experiencing dystocia. The most important predisposing factor to septicemic disease is

hypogammaglobulinemia. Failure of passive transfer of maternal immunoglobulins is directly related to calfhood disease (Boyd, 1972; McGuire et al., 1976; Dardillat et al., 1978; Lumba et al., 1978; Davidson et al., 1981).

The information gained about PCM in the Kansas Flint Hills has merely laid the groundwork for future research. Data from this study on specific infectious and non-infectious agents associated with PCM in this region was also collected and analyzed.

SUMMARY

A systematic necropsy study as to incidence, distribution, general causes and conditions of perinatal calf mortality (PCM) in commercial and purebred operations in the Kansas Flint Hills was conducted over a three year period. Two populations of calf deaths were examined and compared, 155 contract and 82 non-contract. Calf mortalities from 3,934 females expected to calve in contract herds were examined resulting in 7.99% PCM. Incidence was significantly influenced by year ($P \leq .06$) with years 1982 and 1983 being significantly different ($P < .05$). Cow herd size and type of operation also influenced the PCM incidence. The incidence of PCM for the type of operation with respect to cow herd size was significantly different ($P < .05$), with a highly significant negative correlation between incidence of PCM and cow herd size ($P < .001$). There were highly significant differences between calf populations for distribution

by time-of-death (TOD) ($P \leq .0001$) and general causes of PCM ($P \leq .0001$). The contract calf population distribution of PCM was antepartum 6.5%, early partum 7.7%, late partum 28.4%, early postpartum 27.1%, and late postpartum 30.3%. Distribution was significantly influenced by year ($P < .01$) and cow herd size ($P < .0005$). Specific categories of TOD had highly significant differences when compared to general causes of PCM ($P \leq .0001$). General causes of PCM were not influenced by year or type of operation but approached significance with respect to cow herd size ($P \leq .09$). Dystocia was the most frequent condition associated with PCM (25.8%), neonatal weakness was second with a relatively high frequency of 21.9%, and disease complicated starvation was third at 16.8%. Non-infectious conditions were the most prevalant causes of PCM (59.4%) with 55.4% occurring during the parturient period. Infectious causes accounted for 24.5% of all losses with the majority (78.9%) occurring during the late postpartum period. Natal nonviability and neonatal weakness were identified as important conditions associated with PCM with a high frequency of undetermined causes.

REFERENCES

Baker AL, Queensberry JR. Fertility of range beef cattle. J Anim Sci 1944;3:78.

Bar-Anan R. A breeding strategy for reducing perinatal calf mortality in heifer calves. In: Calving Problems and Early Viability of the Calf. Martinus Nijhoff Publ, The Hague, 1979;149-158.

Berg RT. Breeding considerations for minimizing difficult calving. In: Calving Problems and Early Viability of the Calf. Martinus Nijhoff Publ, The Hague, 1979;133-140.

Boyd JW. The relationship between serum immune globulin deficiency and disease in calves: a farm survey. Vet Rec 1972;90:645-649.

Cope GE. Estrus synchronization and reproduction management of beef herds in the south. Proc 14th Ann Conv Am Ass Bov Pract 1982;No 14:104-107.

Dardillat J, Trillat G, Larvor P. Colostrum immunoglobulin concentration in cows: relationship with their calf mortality and with the colostrum quality of their female offspring. Ann Rech Vet 1978;9:375-384.

Davidson JN, Yancy SP, Campbell SG, Warner RG. Relationship between serum immunoglobulin values and incidence of respiratory disease in calves. JAVMA 1981;179:708-710.

Dennis SM. Perinatal mortality of ruminants. Compend Cont Ed Pract Vet 1979;1:S17-S26.

Dennis SM. Investigating perinatal calf mortality. Proc Ann Mtg Soc Theriogenol. Omaha NE, September 10-12, 1980, pp 150-168.

EEC. Calving problems and early viability of the calf. In: Hoffmann B, Mason IL, Schmidt J, ed. Commission of the European Communities, Coordination of Agricultural Research. Martinus Nijhoff Publishers, The Hague, 1979.

Ensminger ME, Galgan MW, Slocum WL. Problems and practices of American cattlemen. Wash Agr Exp Stat Bull 1955;562.

Grunsell GS. Mortality and morbidity in calves. Vet Rec 1956;68:788.

Haughey KG. Meningeal haemorrhage and congestion associated with the perinatal mortality of beef calves. Aust Vet J 1975;51:22-27.

Hubbert WT, et al. Recommendations for standardizing bovine reproductive terms. Cornell Vet 1972;62:216-237.

Khera SS. Fetal and young calf mortality among bovine farmstock in India. Indian J Anim Sci 1981;51:292-302, 425-431, 432-438.

Kincaid CN. Survey of reproduction in S-10 breeding herds. Southern Region Technical Committee S-10, Improvement of Beef Cattle for the Southern Region through Breeding Methods, 1957.

Koger M, Mitchell JS, Kidder RW, Burns WC, Hentges JF, Warnick AC. Factors influencing survival in beef calves. J Anim Sci 1967;26:205.

Laster DB, Gregory KE. Factors influencing peri- and early postnatal calf mortality. J Anim Sci 1973; 37:1092-1097.

Laster DB, Glimp HA, Cundiff LV, Gregory KE. Factors affecting dystocia and the effects of dystocia on subsequent reproduction in beef cattle. J Anim Sci 1973;36:695.

Lumba F, Fumiere I, Tshibangn M, Chauvaux G, Bienfet V. Immunoglobulin transfer to calves and health problems in large bovine units. Ann Rech Vet 1978;9:353-360.

Martin SW, Schwabe CW, Franti CE. Dairy calf mortality rate: characteristics of calf mortality rates in Tulane County, California. Am J Vet Res 1975a;36:1099-1104.

Martin SW, Schwabe CW, Franti CE. Dairy calf mortality rate: influence of meterologic factors on calf mortality rate in Tulane County, California. Am J Vet Res 1975b; 36:1105-1109.

McCormick WC, Southwell BL, Warwick EJ. Factors affecting performance in herds of purebred and grade Hereford cattle. Ga Agr Exp Sta Tech Bull 1956;N.S.5.

McFarlane D. Perinatal lamb losses. 1. An autopsy method for the investigation of perinatal losses. NZ Vet J 1965;13:116-135.

McGuire TC, Pfeiffer NE, Weikel JM, Bartsch RC. Failure of colostral immunoglobulin transfer in calves dying from infectious disease. JAVMA 1976;169:713-718.

Miller RB. A discussion on reproductive failure in cattle. Bovine Pract 1982;17:39-51.

National Academy Science Subcommittee on Prenatal and Postnatal Mortality in Cattle, 1968; Publ no 1685, Washington, DC. pp 1-130.

Olson DP, Bull RC, Kelley KW, Ritter RC, Woodard LF, Everson DD. Effects of maternal nutrition restriction and cold stress on young calves: clinical condition, behavioral reactions, and lesions. Am J Vet Res 1981;42:758-762.

Oxender W, Adams W. Problems associated with calving and neonatal period in beef cattle. In: Mason et al., ed. Calving Problems and Early Viability of the Calf. Commission of the European Communities, Martin Nijhoff Publishers, The Hague, 1979;408-422.

Patterson DJ, Bellows RA, Burfening PJ, Short RE, Carr JB. Incidence and causes of neonatal and postnatal mortality in range cattle; USDA, SEA and Montana State University. J Anim Sci 1979;49(Suppl. 1):325.

Philipsson J. Calving performance and calf mortality. Livest Prod Sci 1976;3:319-331.

Radostits DM, Acres SD. Disease of calves admitted to a large animal clinic in Saskatchewan. Can Vet J 1974;15:82.

Randall GCB. Perinatal mortality: some problems of adaptation at birth. Adv Vet Sci Comp Med 1978;22:53-81.

Rice LE, Wiltbank JN. Factors affecting dystocia in beef cattle. JAVMA 1972;161:1348-1358.

Rice FJ, Woodward RR, Queensberry JR, Willson FS. Fertility of beef cattle raised under range conditions. Mont Exp Sta Bull 1961;561.

Roche JF. Reproductive wastage following artificial insemination of heifers. Vet Rec 1981;109:401-404.

Roy JHB. The calf, 4th ed. Butterworth Inc. 1980;53-66, 369-370, 373-377.

Speicher JA, Hepp RE. Factors associated with calf mortality in Michigan dairy herds. JAVMA 1973;162:463-466.

Stauber EH. Weak calf syndrome: a continuing enigma. JAVMA 1976;168:223-225.

Tayler JC. Beef production in the EEC and the coordination of research by the commission of the European communities. Livest Prod Sci 1976;3:305-318.

Thornberry H. Conclusions from the EEC Seminar on perinatal ill-health in calves. In: Mason et al., ed. Calving Problems and Early Viability of the Calf, Commission of the European Communities, Martin Nijhoff Publishers, The Hague. 1979;487-493.

Wiltbank JN, Warwick EJ, Vernon EH, Priode BM. Factors affecting net calf crop in beef cattle. J Anim Sci 1961; 20:409.

Woodward RR, Clark RT. A study of stillbirths in a herd of range cattle. J Anim Sci 1959;18:85.

Young JS. Breeding patterns in commercial beef herds. Aust Vet J 1968;44:350-356.

Young JS, Blair JM. Perinatal calf losses in a beef herd. Aust Vet J 1974;50:338-344.

TABLE 1 - Time-of-death classification of calves necropsied

Death	Frequency	Percent
Antepartum		
APD 2	24	10.13
APD 3	6	2.53
Partum		
PD 1	1	0.42
PD 2	5	2.11
PD 3	4	1.69
PD 4	12	5.06
PD 5	9	3.40
PD 6	28	11.81
PD 7	4	1.69
Postpartum		
PPD 1	4	1.69
PPD 2	41	17.30
PPD 3	2	0.84
PPD 4	7	2.95
PPD 6	1	0.42
PPD 7	1	0.42
PPD 8	12	5.06
PPD 9	2	0.84
PPD 10	13	5.49
PPD 11	23	9.71
PPD 12	38	16.03
	237	100.00

TABLE 2 - Analysis of time-of-death into five categories

Time-of-Death	Frequency	Percent
Antepartum APD 2 & 3	30	12.66
Early partum PD 1, 2, 3 & 7	14	5.91
Late partum PD 4, 5 & 6	49	20.67
Early postpartum PPD 1 to PPD 7	54	22.78
Late postpartum PPD 8 to PPD 12	90	37.98
	237	100.00

TABLE 3 - Percent distribution of perinatal calf mortality by cause

TOD*	TOD Distribution	Cause*		
		Infectious	Non-Infectious	Unknown
Antepartum	6.5	30.0	10.0	60.0
Partum	36.1	3.6	91.1	5.4
Early postpartum	27.1	7.1	59.5	33.3
Late postpartum	30.3	63.8	31.9	4.3
%		24.5	59.4	16.1

* = Significant difference ($P \leq .0001$)

TOD = time-of-death

TABLE 4 - General conditions of perinatal calf mortality related to time-of-death

TOD & Condition	Calves		
	Contract	Non-contract	Total
Antepartum			30
Abortion	10	19	
Congenital defect	-	1	
Early partum			14
Dystocia	1	2	
Natal nonviability	10	-	
Congenital defect	1	-	
Late partum			49
Birth trauma	4	-	
Dystocia	39	5	
Congenital defect	1	-	
Early postpartum			54
Abortion	-	1	
Acute death	1	1	
Birth trauma	7	-	
Neonatal weakness	30	10	
Congenital defect	4	-	
Late postpartum			90
Acute death	6	12	
CNS dysfunction	2	2	
Complicated starvation	26	20	
Uncomplicated starvation	7	-	
Neonatal weakness	4	5	
Congenital defect	2	4	
	155	82	237

TOD = time-of-death

TABLE 5 - Frequency and time-of-death of the common causes of perinatal calf mortality

		Contract calves				Non-contract calves			
			Cause*				Cause*		
TOD	Condition	f	Inf	N-inf	Unk	f	Inf	N-inf	Unk
Antepartum									
	Abortion	100	30	10	60	95	65	5	30
Early partum									
	Natal nonviability	83	20	60	20	-	-	-	-
	Dystocia	8	-	98	2	100	-	86	14
Late partum									
	Dystocia	89	-	98	2	100	-	86	14
	Birth trauma	9	-	100	-	-	-	-	-
Early postpartum									
	Neonatal weakness	71	9	56	35	83	27	20	53
	Birth trauma	17	-	100	-	-	-	-	-
Late postpartum									
	Complicated starvation	55	88	12	-	47	85	15	-
	Uncomplicated starvation	15	-	100	-	-	-	-	-
	Acute death	13	43	43	14	28	92	8	-
	Neonatal weakness	9	9	56	35	12	27	20	53

* = percent

TOD = time-of-death

TABLE 6 - General causes of perinatal calf mortality by calf population

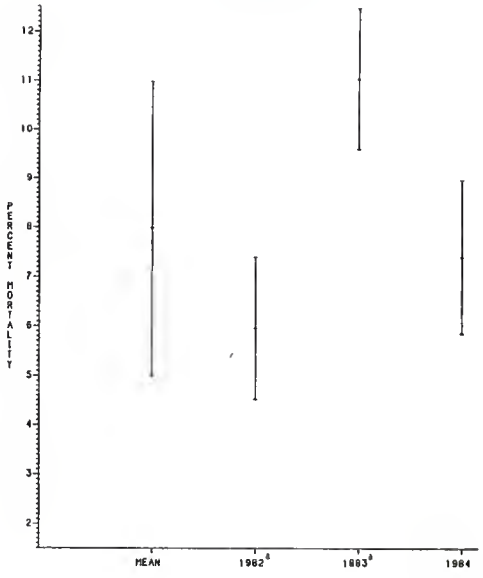
Condition	Calves		Total
	Contract	Non-contract	
Infectious	38	49	87
Non-infectious	92	16	108
Undetermined	25	17	42
	155	82	237

TABLE 7 - Major conditions encounterd in perinatal calf mortality by calf population

Condition	Calves		Total
	Contract	Non-contract	
Abortion	10	20	30
Acute death	7	13	20
Birth trauma	11	-	11
CNS dysfunction	2	2	4
Complicated starvation	26	20	46
Uncomplicated starvation	7	-	7
Dystocia	40	7	47
Natal nonviability	10	-	10
Neonatal weakness	34	15	49
Congenital defect	8	5	13
	155	82	237

Fig 1 - Regional incidence of PCM with respect to year. Mortality incidence between years was significantly different ($P < 0.05$).

Fig 2 - Regional PCM incidence with respect to type of operation (adjusted for cow herd size). Mortality incidence was significantly lower ($P < 0.06$) for purebred than commercial herds.


PERCENT MORTALITY
12
11
10
9
8
7
6
5
4
3
2
MEAN
1982[a]
1983[a]
1984

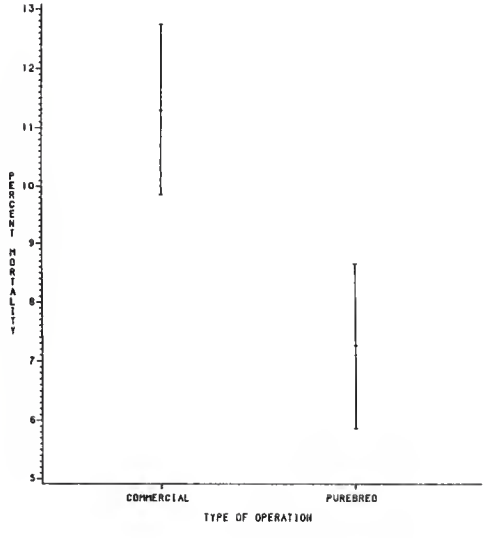

PERCENT MORTALITY
13
12
11
10
9
8
7
6
5
COMMERCIAL
PUREBRED
TYPE OF OPERATION

Fig 3 - Incidence of calf mortality with respect to size and type of cow-calf operation. Mortality incidence with respect to type and size of operation was significantly different ($P < .05$). Change in mortality incidence with respect to cow herd size was significantly different within commercial operations ($P < .001$).

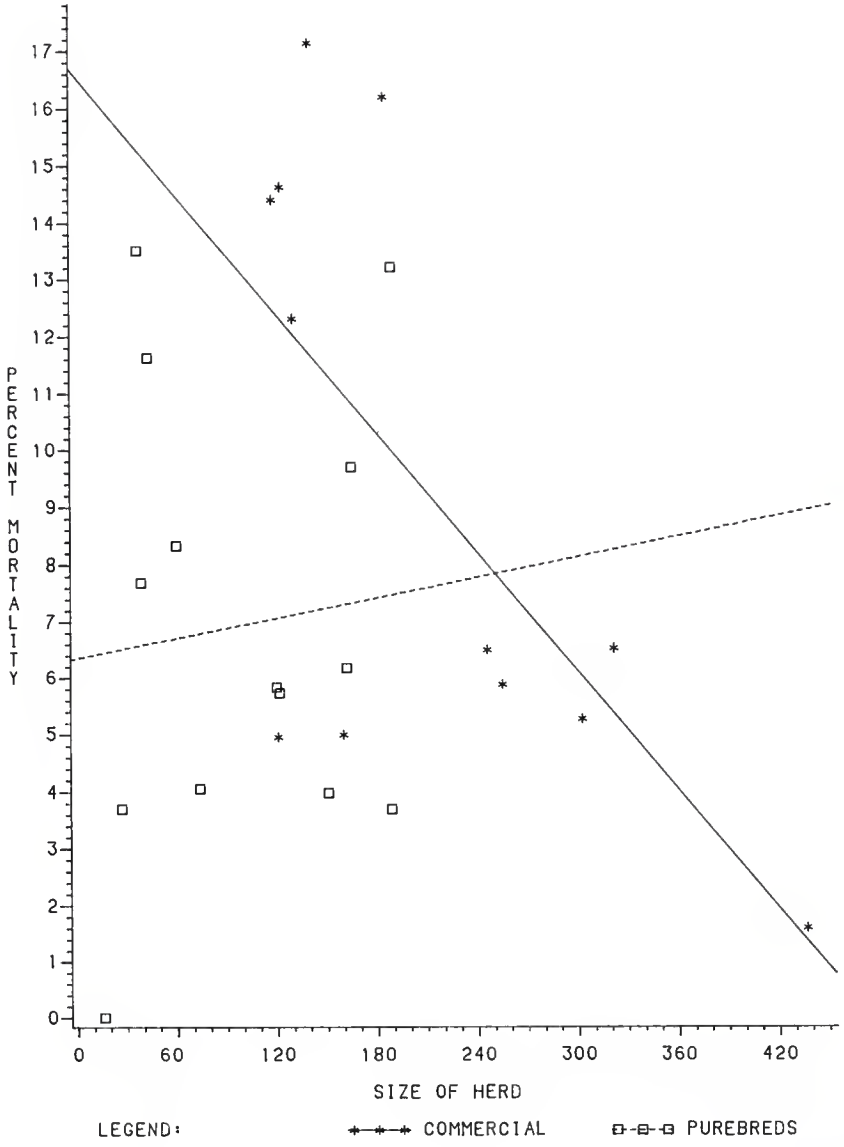
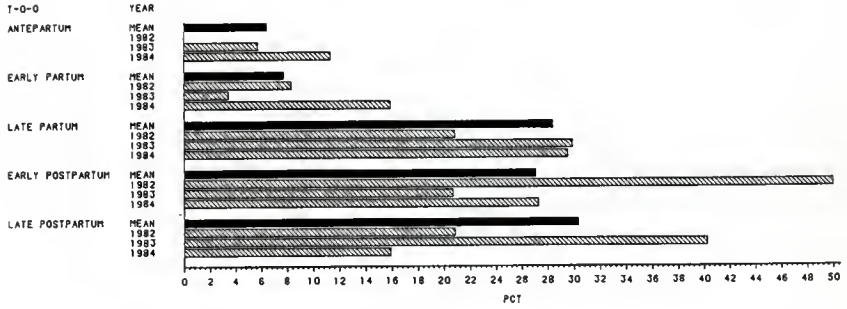

PERCENT MORTALITY
0
1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
0
60
120
180
240
300
360
420
SIZE OF HERD
LEGEND:
COMMERCIAL
PUREBREDS

Fig 4 - Regional distribution of PCM -- yearly and the three year average. Mortality distribution was significantly different between years ($P \leq 0.01$).

Fig 5 - Regional distribution of PCM with respect to cow herd size. Mortality distribution was significantly different between size of operation ($P < 0.0005$).


T-O-O
YEAR
ANTEPARTUM
MEAN
1982
1983
1984
EARLY PARTUM
MEAN
1982
1983
1984
LATE PARTUM
MEAN
1982
1983
1984
EARLY POSTPARTUM
MEAN
1982
1983
1984
LATE POSTPARTUM
MEAN
1982
1983
1984
0 2 4 6 8 10 12 14 16 18 20 22 24 26 28 30 32 34 36 38 40 42 44 46 48 50
PCT

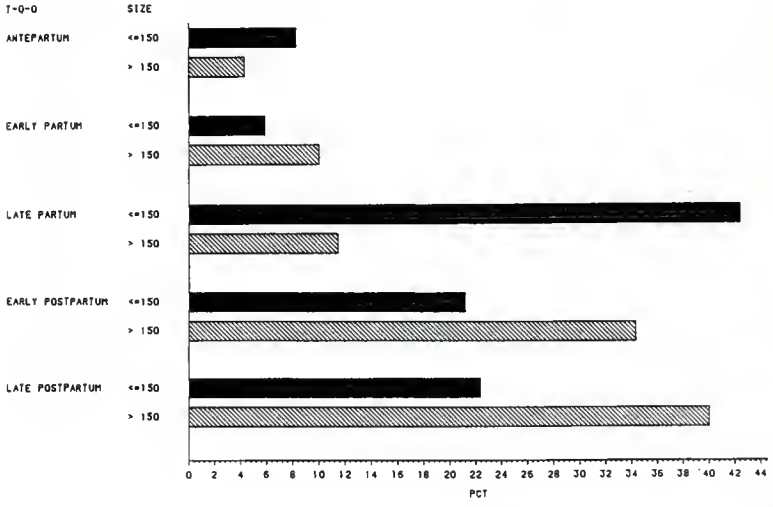
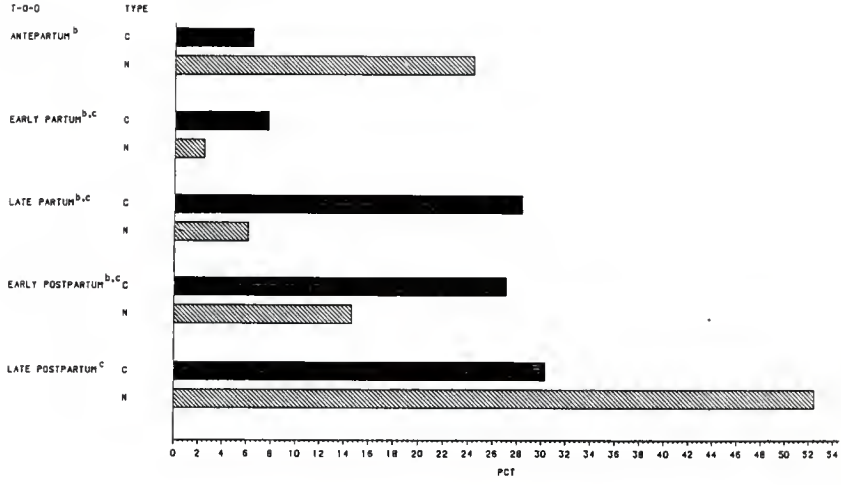

T-O-O
SIZE
ANTEPARTUM
<=150
> 150
EARLY PARTUM
<=150
> 150
LATE PARTUM
<=150
> 150
EARLY POSTPARTUM
<=150
> 150
LATE POSTPARTUM
<=150
> 150
0 2 4 6 8 10 12 14 16 18 20 22 24 26 28 30 32 34 36 38 40 42 44
PCT

Fig 6 - Regional distribution of PCM with respect to calf population. Mortality incidence was significantly different ($P \leq 0.0001$) between calf populations and time-of-death (a,b,c).
C = contract calf population; N = non-contract calves.

Fig 7 - General causes of PCM with respect to calf populations. Infectious and non-infectious causes were significantly different between the two calf populations ($P \leq 0.0001$) and the non-infectious and unknown causes ($P < 0.001$).


T-O-O
TYPE
ANTEPARTUM b
C
N
EARLY PARTUM b,c
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LATE PARTUM b,c
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EARLY POSTPARTUM b,c
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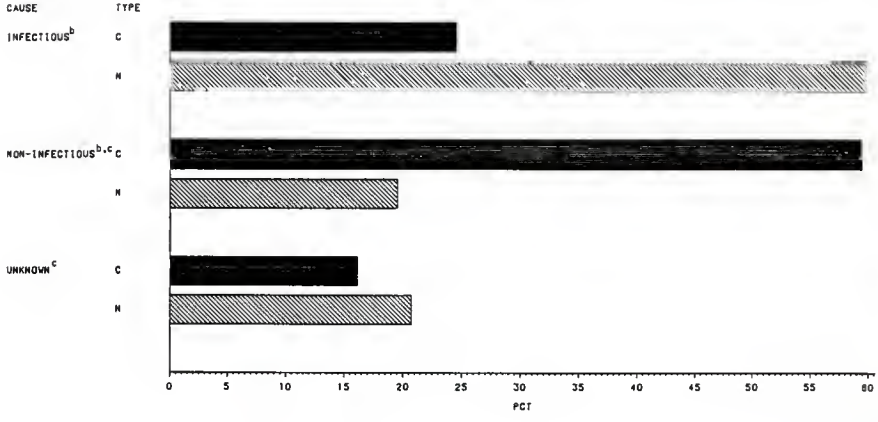
0 2 4 6 8 10 12 14 16 18 20 22 24 26 28 30 32 34 36 38 40 42 44 46 48 50 52 54
PCT


CAUSE
TYPE
INFECTIOUS b
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0 5 10 15 20 25 30 35 40 45 50 55 60
PCT

III. PERINATAL CALF MORTALITY IN THE KANSAS FLINT HILLS: INFECTIOUS CAUSES

INTRODUCTION

Infectious causes of perinatal calf mortality (PCM) play a significant role in this complex problem and have received considerable attention, especially in specific problem areas such as abortion, enteritis and pneumonia. Perinatal infections may be congenital or acquired after birth. The outcome of fetal infection depends on the stage of gestation when infected: resorption, abortion, dysmaturity, premature births, developmental defects, neonatal weakness, birth of viable carriers, or normal neonates. The earlier in gestation infection becomes established, the more likely the outcome of fetal death and abortion. Infection later in gestation has a greater chance of resulting in normal parturition and a surviving neonate (Dennis, 1980; 1981).

The only lesion common to all forms of fetal infections is placentitis. The fetus may be invaded from the placenta directly via the umbilical vein or indirectly by inhalation and ingestion of infected amniotic fluid or via the skin (Dennis, 1979). The common etiological causes of abortion may vary from region to region (Hubbert et al., 1973; Kirkbride et al., 1973; Kirkbride, 1979; K.S.D.L., 1981; Moojen et al., 1983). Etiological diagnosis of abortion is often difficult and the diagnostic success of laboratories around the world is only 30 to 40% (Kirkbride, 1984). Fetal

serology has increased the percentage of abortions diagnosed (Horner et al., 1973; Sawyer et al., 1973; Ellis et al., 1978; Moojen et al., 1983).

Invasion of the neonate after birth by infections agents is common and the outcome is determined not only by the type of pathogen but also by the calf's interaction with its environment. Failure of passive transfer of maternal antibodies is one environmental factor reported to be directly related to various types of neonatal disease (Boyd, 1972; McGuire et al., 1976; Dardillat et al., 1978; Lumba et al., 1978; Davidson et al., 1981). After parturition, the calf has high levels of circulating corticosteroids that inhibit its inherent defenses rendering it at risk from agammaglobulinemia, lymphopenia and decreased phagocytic activity (Osburn et al., 1974). To be adequately protected, a newborn calf must ingest maternal antibodies within the first 24 hours after birth, maximum colostral absorption occurs during the first 6 to 8 hours (Dennis, 1981). The potential growth of a calf is positively correlated with its globulin level at day 8 (Thornberry, 1979).

Neonatal infections during the first week of life tend to be septicemic or toxemic. These infections progress and tend to localize in various organs such as joints, brain, lung, kidney, liver and intestines. Transmission of infection may be by contamination of the umbilical cord or by inhalation or ingestion (Dennis, 1980). The major infectious calf disease in beef cattle continues to be enteritis

(Oxender and Adams, 1979) and numerous bacteria and viruses have been implicated (Acres et al., 1975). The number of viruses associated with neonatal infections continues to rise in direct proportion to increased utilization of diagnostic facilities and more refined laboratory techniques (Evermann, 1979).

Enterotoxic E. coli appears to be the commonest single agent causing diarrhea (Dennis, 1981). Torres-Medina (1984) reported that non-enterotoxigenic E. coli caused severe diarrhea and villous atrophy when combined with rotavirus.

This paper reports the infections diagnosed in calves in the Kansas Flint Hills during a 3-year investigation into the causes of perinatal calf mortality.

MATERIALS AND METHODS

The general procedures in this investigation were described in Part II. All calves were necropsied and the time-of-death (TOD) assessed (McFarlane, 1965). The deaths were divided into five categories: antepartum (APD), early partum (EPD), late partum (LPD), early postpartum (EPPD), and late postpartum (LPPD).

Diagnostic techniques employed included darkfield microscopy, bacteriology, immunofluoresence, serology, enzyme-linked immunosorbent assay (ELISA), virus isolation, and fetal immunoglobin (Ig) content and over the 3-years, a laboratory diagnostic protocol was developed. All tests except Ig and darkfield microscopy, were performed by the Kansas State Diagnostic Laboratory.

Darkfield Examination

Darkfield examination for Leptospira, Campylobacter, Aspergillus, and Trichomonas species were performed on fetal fluid obtained from abdominal, thoracic and pericardial cavities, abomasum, and occasionally maternal uterine fluid. All samples were examined under high dry power (400X). Positive identification was made on the pathogen's characteristic morphology and movements.

Bacteriological Examination

Specimens submitted for culture included swabs of thoracic and abdominal cavity fluids, abomasal and small intestinal contents, and tissues with gross lesions, and various tissues. Swabs were streaked directely onto 5% sheep blood and MacConkey's agar plates. Tissue samples were trimmed, swabed and streaked onto 5% sheep blood and MacConkey's agar plates.

Plates were incubated in 5½% CO_2 at 37 C for 5 days. When Clostridium spp. were suspected, plates were incubated under anaerobic conditions at 37 C for the same period. Bacteria isolated were identified by standard differential techniques.

Immunofluorescence

Tissues submitted for fluorescent antibody (FA) testing included lung, liver, spleen, kidney, small intestine, placenta, and occasionally brain. FA tests for IBR, BVD and

PI_3 were routinely performed. Tests for other pathogens such as coronavirus were performed when suspected. All tissues were cut by a cryostat into sections, 6 to 8 microns thick, processed and stained with specific fluorescein conjugated antibody by techniques described for diagnosing IBR, BVD and PI_3 (Brown et al., 1968), and coronavirus (Mebus et al., 1975).

Serological Examination

Postmortem fetal blood samples were obtained from axillary blood vessels during the initial part of necropsy. Blood was centrifuged at 2,800 rpm, serum decanted and split into two equal volumes, and stored at -70 C for later testing. When sufficient number of serum samples had been collected, a sample from each calf was thawed and submitted for serological testing for antibody to IBR, BVD, PI_3, Leptospira pomona, L. hardjo, L. grippotyphosa, L. icterohaemorrhagiae, L. canicola, Brucella abortus, Haemophilus somnus, and bluetongue virus.

Antibodies specific for IBR, BVD, PI_3 were determined by microtiter serum neutralization tests described by Carbey et al. (1971). The cell cultures were embryonic bovine kidney and the appropriate viral suspensions contained 250 median tissue culture infectious doses ($TCID_{50}$). Dilutions started at 1:2 and were carried out to 1:256. In nonsuckled serum samples titers of 1:2 or greater were considered significant. A standard agar gel immunodiffusion test was employed for detecting bluetongue virus antibodies.

A standard microscopic agglutination test for antibodies to the 5 most common serovars of Leptospira interrogans found in cattle was used with fresh live antigens and the samples screened at 1:50 dilution. The standard card agglutination test was used to test for antibodies to Brucella abortus.

Haemophilus somnus antibody was detected by a micro-agglutination test using a H. somnus MA stock antigen[a]. Negative controls were determined at less than or equal to 1:8. Titers of 1:8 or greater from unsuckled calves were considered significant.

Enzyme-linked Immunosorbent Assay

ELISA tests were employed in the diagnostic work-up of calves classified as late postpartum deaths. A commercial Rotazyme[b] diagnostic kit for detecting calf rotavirus was used and the manufacturer's testing procedure was followed. In suspected cases of enterotoxigenic E. coli infections a monoclonal antibody ELISA for K-99 antigen was used (Mills and Tietze, 1984).

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[a]Phillips Roxane Inc., 2621 North Belt Highway, St. Joseph MO 64502

[b]Rotazyme by Abbott Laboratories, North Chicago IL 60064

Virus Isolation

Tissues routinely submitted for virus isolation were lung, liver, spleen and kidney. Tissues were cut into 3 mm square pieces, homogenized and stored at -70 C. Samples were thawed and passaged on embryonic bovine kidney cell cultures in 25 square cm flasks and incubated at 37 C. Two more passages were made at weekly intervals. If no cell culture changes were noticed after the third passage, a FA test was performed to detect viral particles for BVD. If cell culture changes occurred, an attempt to identify the virus particles was made by FA techniques or electron microscopy. Unidentified viruses were sent to the National Animal Disease Center, Ames, IA for identification.

Fetal Serum Immunoglobulin

Total fetal serum Ig content was semiquantitatively derived by using the sodium sulfite precipitation test described by Pfieffer and McGuire (1977). Serum was extracted and stored as previously described. When sufficient sample numbers were obtained, they were thawed for testing. At the end of 1 hour, at room temperature, test results were recorded at less then 5 mg/ml, 5-15 mg/ml, or greater then 15 mg/ml.

RESULTS

A total of 237 calves were necropsied and laboratory diagnostic tests were performed on most, especially during

the last two years of the investigation. Fetal serum samples found to be unfit for serological testing were unfit also for testing for Ig content.

Incidence and Distribution of Infectious PCM

Infectious causes of PCM in the Flint Hills of Kansas were responsible for almost a quarter (24.5%) of all calves examined from herds agreeing to submitt all perinatal losses. The cause of death could not be determined in 16.1% of the calves but a high percent were considered to be infectious on histopathologic examination.

The incidence when distributed between the 5 classes of TOD provided a more useful approach for identifying where problem areas actually lie. Ninety percent of calves dying in the antepartum category died from infectious or undetermined causes (30% and 60%, respectively). In the early partum category, 33.3% died of infectious or undetermined causes, both with an equal percentage. No infectious causes were found in calves dying in the late partum or dystocia category. Early postpartum deaths were 40.5% infectious or undetermined, with undetermined causes accounting for 82%. A high percent (68.1%) of calves dying in the late postpartum category died of infectious or undetermined causes, with 94% being infectious.

Bacteriologic Results

Over the 3-year period, culturing was done on 61% of all calves necropsied and significant isolates were obtained

74.5% of the time. Serological testing for antibodies of various bacteria was done on 18% of all calves, and 100% of the calves examined during the 1984 calving season. Dark-field examination of fetal fluids was done on 12% of all calves, mostly fetal deaths.

The frequency of bacteria cultured, with respect to TOD, from all calves examined over the three years is summarized in Table 1. In calves where an infectious cause of death was determined, the pathogens incriminated with the disease are given in Table 2. Examining the frequency of pathogenic bacteria in relation to TOD was helpful in assessing when the various bacteria most commonly cause problems (Table 3). Overall, the incidence of pathogenic bacteria found in the antepartum, early postpartum, and late postpartum TOD categories were 15.5%, 8.6% and 75.9%, respectively.

Viral Results

One or more FA tests for viruses were performed on 38% of all calves necropsied over the three year period, a total of 218 with approximately 6% positive. Virus isolation was performed on 20% of all calves (45) with positive isolation occurring 2 (4%). Serological examination for viral antibodies was also performed in the same frequency as for bacterial antibodies. The majority of the viral diagnostic tests were performed during the 1984 calving season. All positive viral isolations, FA identification and serologic

results were considered significant in calves with lesions or histories suggestive of infectious disease (Tables 2 and 3). ELISA for rotavirus was performed 15 times and 47% were either positive or suspicious for rotavirus.

Dual Infections

Due to the number of diagnostic tests performed, identification of calves infected with multiple agents was possible (Table 4). Dual infection was identified in 6 calves (2.5%), approximately 8% of all calves dying from infectious causes. BVD and Haemophilus somnus were the pathogens associated most frequently with dual infection. This combination accounted for 22% of the infectious causes of neonatal weakness. BVD and Leptospira spp. in combination were incriminated as a dual cause of abortion.

Individually BVD was most frequently found in combination. Haemophilus somnus, rotavirus and E. coli were next most frequent followed by C. perfringens, Leptospira spp., and Salmonella spp. The various combinations of pathogenic agents found in dual infection with respect to the specific condition and associated TOD are summarized in Table 4.

DISCUSSION

A laboratory testing protocol was developed to determine whether diagnostic efficiency is affected by the number and types of tests performed, and if so, how much effect and which tests had the most influence. The testing

protocol was developed in three phases with each phase beginning and ending yearly. Phase I, the first year approach, called for a minimum number of diagnostic tests if there was sufficient evidence, history or gross lesions, to warrant their use. Necropsy floor diagnostic procedures were employed such as tissue and fluid smears stained Gram, modified acid-fast, and Wright (Dif-Quik) techniques, and darkfield examination of body fluids. The aim was to simulate a practice situation with limited resources and diagnostic tests available.

With Phase II in the second year, there was a moderate increase in number and breath of the laboratory tests. Two to four tests were utilized to narrow the differential diagnoses, primarily bacteriology, serology, and immunofluoresence. Serological and FA testing for IBR, BVD, PI_3, rota, and corona viruses were performed at times. Additional tests included darkfield examination, virus isolation, and fetal Ig levels. The aims were to simulate a common diagnostic laboratory situation by utilizing a moderate number of tests to increase diagnostic efficiency but conserve costs.

In the final year, the laboratory diagnostic protocol (Phase III) made maximum use of available tests (Appendix 6). The following tests were performed on calves that had not suckled: bacterial culture of thoracic, abdominal and abomasal swabs; FA tests for IBR, BVD and PI_3 viruses; serology for IBR, BVD, PI_3, Leptospira serovars (canicola,

grippotyphosa, hardjo, icterohaemorrhagiae, and pomona), B. abortus, H. somnus, and bluetongue virus; darkfield examination for campylobacteria, fungi, and trichomonads; and fetal serum Ig levels. For calves that suckled, the same tests were performed except for small intestinal contents replacing abomasal contents, FA test for coronavirus, ELISA for rotavirus, and an occasional ELISA for E. coli K-99 antigen. The aim of phase III was to maximize diagnostic efficiency of PCM by increased laboratory testing.

Increasing the number of laboratory tests performed on each calf necropsied lowered the percentage of undetermined causes of PCM each year. During the first year with minimal diagnostic laboratory support, 29% of calves died of undetermined causes; employing a moderate number of diagnostic tests during the second year, 15% undetermined causes, and the third year with maximum diagnostic support, only 11% were undetermined. Reduction in 29% undiagnosed in the first year with minimal laboratory testing by almost a half to 15% during the second year by using moderate laboratory support is highly significant. The reduction of unknown diagnoses by 4%, between the second and third year, may not be sufficient enough to economically warrant routine use of maximum laboratory testing. The numbers were insufficient to determine whether a statistically significant difference existed between phase II and III. It is concluded, however, that if an individual case or herd demanded a high degree of

diagnostic efficiency, an increased number of laboratory tests including bacteriology, FA, ELISA, serology, dark-field, and virus isolation, will be beneficial.

Identifying the type of infectious agent causing the PCM was highly influenced by the number of laboratory tests performed. During the first two years bacteria accounted for 82% and viruses for 18% of the infectious PCM. During the third year viral agents were identified 63% of the time as primary causes of PCM, whereas, bacteria accounted for only 38%. Identifying an accurate incidence of viral causes of PCM requires a broader range of diagnostic tests.

All abortions examined were sporadic. Establishing a diagnosis in sporadic abortion is more difficult than with epidemic abortion (Kirkbride, 1979). Leptospiral abortions were the most common diagnosed, one was a dual infection with BVD. IBR was the major viral cause of abortion. Moojen (1983) reported that Leptospira sp. and IBR were the most commonly isolated or demonstrated causes of abortion in Michigan. In this study, H. somnus, PI-3 and BVD followed leptospirosis and IBR in incidence. IBR and BVD were the only causes of natal nonviability detected.

Bovine viral diarrhea virus was the viral agent associated with the widest range of perinatal infections. This is similar to the findings of Evermann (1979). BVD was involved with: abortion (12%), natal nonviability (50%), neonatal weakness (57%), pneumonia (25%), and enteritis (4%).

H. somnus was the bacterial agent associated with the widest range of perinatal infections; abortion (12%), pneumonia (50%), neonatal weakness (29%), and omphaloarthritis (50%).

E. coli continues to be the most common agent associated with enteritis. It was also the agent most commonly associated with septicemic conditions. Rotavirus was the most prevalant viral enteropathogen encountered. This finding agrees with Acres et al., (1975) but conflicts with that of House (1978) and Evermann (1979). Coronavirus was not isolated as a cause of enteritis in this study, but this was probably due to lack of samples submitted for coronavirus determination.

Acute death in large healthy calves was identified as a major cause of infectious PCM. Clostridium perfringens was isolated in 58% of these late postpartum deaths. The acute death and characteristic lesions result from the enterotoxin produced by the clostridia. Bovine enterovirus type 2 was isolated as a cause of acute death during the late postpartum period.

Over the three years, the efficiency of the five categories of laboratory diagnostic tests were examined and bacterial culturing was found to be the most efficient. Due to the ease of performing this technique and without the need of expensive materials, it is recommended that routine culturing be done on all calves suspected of dying from an infectious cause.

Serology using a single sample of postmortem serum or body fluid proved to be an efficient diagnostic procedure. Of all the serological tests performed, 14% gave a positive titer with IBR, BVD, PI-3 and H. somnus comprising 96% of all the positive tests. The frequency of positive titers arising from one of the four serological tests was high (36%). Serological testing for Leptospira serovars and B. abortus was not useful in this study. Perinatal antibodies to the bluetongue virus were found only in two calves that had nursed. If they were present in presuckled calf serum they were not in sufficient concentration to be detected by agar-gel immunodiffusion. The majority of serological tests were performed on calves necropsied during the final year. There was a highly significant relationship between TOD and serological results during that year.

The highest percent of positive serological tests (30%) was found in the early postpartum period, most commonly associated with neonatal weakness. Serology was also an important diagnostic procedure for identifying dual infections. The presence of specific detectable antibodies in samples tested from calves dying before ingesting colostrum suggest that sublethal in utero infection occurs and plays an important role in the calf viability.

Attention must be given as to the condition of serum samples. Samples from severely autolytic calves will give false positives. The results from this study indicate that single sample tests for IBR, PI-3, BVD and H. somnus are

beneficial for an etiological diagnosis in calf deaths occurring during the antepartum, early partum and early postpartum TOD categories.

Darkfield examination was 21% efficient in identifying infectious agents. It was an effective diagnostic tool for diagnosing infectious abortion as 35% of the infectious causes of abortion were diagnosed by this method.

FA and ELISA tests were beneficial in the etiological diagnosis of postpartum infectious conditions, specifically pneumonia and enteritis. ELISA for rotavirus, combined with bacteriologic culturing, proved to be effective in determining single and dual causes of enteritis. Both operations are fairly simple and can be performed in most practitioners' laboratories. More information on multiple agent infections and interactions are needed to better understand and prevent neonatal calf diarrhea.

Viral isolation had a low efficiency in this perinatal calf mortality study. Postmortem autolysis and viral lability were major factors for the low efficiency. Virus isolation, however, proved to be an important research tool as one of the two viruses isolated was bovine enterovirus type 2, an uncommon cause of acute death. Routine use of viral isolation for diagnosing PCM is questionable due to amount of time and costs involved. Serologic and FA testing for viral agents is effective, quicker and more economical.

SUMMARY

Infectious causes encountered during a 3-year investigation of perinatal calf mortality of beef cattle in the Flint Hills of Kansas are reported. Incidence of infectious and undetermined causes of PCM was 24.5% and 16.1%, respectively. Systematic necropsy examination of 237 dead calves (85 of which specific infectious causes could be determined) were performed. They were classified by time-of-death (TOD) and various diagnostic tests performed. The increased use of laboratory tests decreased the incidence of undetermined diagnoses and also increased the percent of viral causes of PCM diagnosed. Bacterial culturing was the most efficient diagnostic tool used followed by single sample postmortem serology of the calf, darkfield examination, immunofluoresence and ELISA testing, and virus isolation. Serologic results were significantly related to TOD ($P \leq .0001$). Leptospirosis and IBR were the major causes of abortion; *E. coli* and rotavirus were the major causes of enteritis; *E. coli* was the major cause of septicemia; and *Clostridium perfringens* was the major cause of acute infectious deaths. BVD and *H. somnus* were the most common causes of infectious neonatal weakness and were also associated with a wide range of neonatal infectious conditions. Dual infections were found in 2.5% of all calf deaths and 8% of all infectious conditions.

REFERENCES

Acres SD, Laing CJ, Saunders JR, Radostits OM. Acute undifferentiated neonatal diarrhea in beef calves. 1. Occurrence and distribution of infectious agents. Can J Comp Med 1975;39:116-132.

Boyd JW. The relationship between serum immune globulin deficiency and disease in calves: a farm survey. Vet Rec 1972;90:645-649.

Brown CL, Jenney EW, Lee LR. Fluorescent antibody procedures for bovine viruses. Rep 72nd Ann Mtg US Lvstk San Assoc 1968; 470-477.

Carbrey EA, et al. Recommended standard laboratory techniques for diagnosis of IBR, BVD, PI-3. Proc Ann Gen Mtg Am Assoc Vet Lab Diag, Oklahoma City, 1971.

Dardillat J, Trillat G, Larvor P. Colostrum immunoglobulin concentration in cows: relationship with their calf mortality and with the colostrum quality of their female offspring. Ann Rech Vet 1978;9:375-384.

Davidson JN, Yancy SP, Campbell SG, Warner RG. Relationship between serum immunoglobulin values and incidence of respiratory disease in calves. JAVMA 1981;179:708-710.

Dennis SM. Perinatal mortality of ruminants. Compend Cont Ed Pract Vet 1979;1:S17-S26.

Dennis SM. Investigating perinatal calf mortality. Proc Ann Mtg Soc Theriogenol. Omaha NE, September 10-12, 1980, pp 150-168.

Dennis SM. Low viability of calves at birth. Vet Ann 1981;21:63-73.

Ellis WA, Logan EF, O'Brien JJ. Serum immunoglobins in aborted and non-aborted bovine foetuses. Clin Exp Immunol 1978;33:136-141.

Evermann JF. Calfhood morbidity and mortality in the Northwestern United States 1977-1979. Proc 22nd Ann Mtg Am Assoc Vet Lab Diag 1979;379-394.

Horner GW, Johnson RH, Dennett DP, Lane WR. A serological study of bovine foetal immunoglobulins. Aust Vet J 1973;49:325-329.

House JA. Economic impact of rotavirus and other neonatal disease agents of animals. JAVMA 1978;173:573-576.

Hubbert WT, Booth GD, Bolton WD, Dunne HW, McEntee K, Smith RE, Tourtellotte ME. Bovine abortions in five Northeastern states, 1960-1970; Evaluation of diagnostic laboratory data. Cornell Vet 1973;63:291-316.

Kansas State Diagnostic Laboratory. Annual Report of Incidence of Infectious causes of Disease in Kansas. 1981.

Kirkbride CA. Abortive diseases of cattle: their significance and prevalence. Vet Med/SAC 1979;74:1151-1155.

Kirkbride CA. Laboratory diagnosis of abortion in food animals. Am Assoc Vet Lab Diag Inc Publ, Wisconsin, 1984, 3.

Kirkbride CA, Bicknell EJ, Reed DE, Robl MG, Knudtson WU, Wohlgemuth K. A diagnostic survey of bovine abortion and stillbirth in the northern plains states. JAVMA 1973; 162:556-560.

Lumba F, Fumiere I, Tshibangn M, Chauvaux G, Bienfet V. Immunoglobulin transfer to calves and health problems in large bovine units. Ann Rech Vet 1978;9:353-360.

McFarlane D. Perinatal lamb losses. 1. An autopsy method for the investigation of perinatal losses. NZ Vet J 1965;13:116-135.

McGuire TC, Pfeiffer NE, Weikel JM, Bartsch RC. Failure of colostral immunoglobulin transfer in calves dying from infectious disease. JAVMA 1976;169:713-718.

Mebus CA, Newman LE, Stair EL, Jr. Scanning electron, light and immunofluorescent microscopy of intestine of gnotobiotic calf infected with diarrhea coronavirus. Am J Vet Res 1977;36:1-19.

Mills KW, Tietze KL. Monoclonal antibody enzyme-linked immunosorbent assay for identification of K99-positive Escherichia coli isolates from calves. J Clin Microbiol 1984;19:498-501.

Moojen V, Roberts AW, Carter GR. Microbial causes of bovine abortion in Michigan. Vet Med/SAC January 1983; 78:102-106.

Osburn BI, Stabenfeldt GH, Adams AA, Trees C, Sawyer M. Perinatal immunity in calves. JAVMA 1974;164:295-298.

Oxender W, Adams W. Problems associated with calving and neonatal period in beef cattle. In: Mason et al., ed. Calving Problems and Early Viability of the Calf. Commission of the European Communities, Martin Nijhoff Publishers, The Hague, 1979;408-422.

Pfeiffer NE, McGuire TC. A sodium sulfite-precipitation test for assessment of colostral immunoglobulin transfer to calves. JAVMA 1977;170:809-811.

Sawyer M, Osburn BI, Knight HD, Kendrick JW. A quantitative serologic assay for diagnosing congenital infections of cattle. Am J Vet Res 1973;34:1281-1284.

Thornberry H. Conclusions from the EEC Seminar on perinatal ill-health in calves. In: Mason et al., ed. Calving Problems and Early Viability of the Calf, Commission of the European Communities, Martin Nijhoff Publishers, The Hague. 1979;487-493.

Torres-Medina A. Effect of combined rotavirus and E. coli in neonatal gnotobiotic calves. Am J Vet Res 1984;45:643-651.

TABLE 1 - Bacteria isolated from calves relative to time-of-death

Bacteria	APD*	EPD	LPD	EPPD	LPPD	Total
Escherichia coli	4	3	3	10	49	69
α Streptococcus sp	3	1	1	6	8	19
Corynebacterium pyogenes	1	1	-	2	3	7
Clostridium perfringens	-	-	-	1	7	8
Proteus sp	3	-	-	-	3	6
Haemophilus somnus	1	-	-	1	2	4
Pasteurella hemolytica	-	-	-	-	4	4
Pasteurella multocida	-	1	-	1	1	3
Salmonella sp	-	-	-	-	2	2
Pseudomonas aeruginosa	-	-	-	-	2	2
Staphylococcus aureus	1	-	-	1	-	2
Staphylococcus sp	-	-	-	-	2	2
Clostridium sordelli	-	-	-	-	1	1
Klebsiella sp	-	-	-	-	1	1
Streptococcus suis II	-	-	1	-	-	1
Branhamella sp	-	-	-	1	-	1
Micrococcus sp	-	-	-	1	-	1
Total	13	6	5	24	85	133

*APD = antepartum death
EPD = early partum death
LPD = late partum death
EPPD = early postpartum death
LPPD = late postpartum death

TABLE 2 - Causes of infectious conditions associated with perinatal calf mortality

Condition	N	Bacterial and Mycotic	N	%	Viral	N	%
Abortion	17	Leptospira sp	5	29.4	IBR	3	17.6
		Haemophilus somnus	2	11.8	BVD	2	11.8
		Aspergillus sp	1	5.9	PI-3	2	11.8
		Corynebacterium pyogenes	1	5.9	Chlamydia sp	1	5.9
Acute death	12	Clostridium perfringens	7	58.3	Bovine enterovirus type 2	1	8.3
		Escherichia coli	3	25.0			
		Pasteurella hemolytica	1	8.3			
Enteritis	25	E. coli	17	68.0	Rotavirus	4	16.0
		Salmonella sp	2	8.0	BVD	1	4.0
		Cl. perfringens	1	4.0			
Neonatal weakness	7	H. somnus	2	28.6	BVD	4	57.1
					Rotavirus	1	14.3
Septicemia	7	E. coli	5	71.4			
		C. pyogenes	1	14.3			
		Staphylococcus aureus	1	14.3			
Pneumonia	4	H. somnus	2	50.0	BVD	1	25.0
		P. hemolytica	1	25.0			
Omphaloarthritis	4	H. somnus	2	50.0			
		C. pyogenes	1	25.0			
		Cl. sordelli	1	25.0			
Natal nonviability	2				BVD	1	50.0
					IBR	1	50.0
Neurologic disorder	2	E. coli	2	100.0			
Total	80		58	72.5		22	27.5

TABLE 3 - Isolation of infectious agents from calves relative to the time-of-death

	APD	EPD	LPD	EPPD	LPPD	Total
Bacterial and Mycotic						
Escherichia coli	-	-	-	-	27	27
Haemophilus somnus	2	-	-	4	2	8
Clostridium perfringens	-	-	-	1	7	8
Leptospira sp	5	-	-	-	-	5
Corynebacterium pyogenes	1	-	-	-	2	3
Pasteurella hemolytica	-	-	-	-	2	2
Salmonella sp	-	-	-	-	2	2
Aspergillus sp	1	-	-	-	-	1
Clostridium sordelli	-	-	-	-	1	1
Staphylococcus aureus	-	-	-	-	1	1
Subtotal	9	0	0	5	44	58
Viral						
BVD virus	2	1	-	2	4	9
Rotavirus	-	-	-	1	4	5
IBR virus	3	1	-	-	-	4
PI-3 virus	2	-	-	-	-	2
Bovine enterovirus type 2	-	-	-	-	1	1
Chlamydia sp	1	-	-	-	-	1
Subtotal	8	2	0	3	9	22
Total	17	2	0	8	53	80

APD = antepartum death
EPD = early partum death
LPD - late partum death
EPPD = early postpartum death
LPPD = late postpartum death

TABLE 4 - Dual infections associated with perinatal calf mortality

Time-of-death	Condition	(N)	Infectious agents	% dual infections
Antepartum	Abortion	1	*Leptospira* sp & BVD virus	16.7
Early partum	-		-	-
Late partum	-		-	-
Early postpartum	Neonatal weakness	2	*Haemophilus somnus* & BVD virus	33.3
Late postpartum	Enteritis	3	*Escherichia coli* & *Clostridium perfringens*	16.7
			E. coli & rotavirus	16.7
			Salmonella sp & rotavirus	16.7

IV. PERINATAL CALF MORTALITY IN THE KANSAS FLINT HILLS: NON-INFECTIOUS FACTORS

INTRODUCTION

In contrast to infectious causes of perinatal calf mortality, non-infectious causes have received little attention. This is paradoxical as systematic investigations of perinatal calf mortality have incriminated the majority of losses as non-infectious (Young and Blair, 1974; Patterson, 1979). Non-infectious factors can also predispose calves to infections by minor pathogens that would otherwise be innocuous.

The incidence of perinatal calf mortality (PCM) in beef cattle is reported to be 3.5% to 25% of all females diagnosed as being pregnant with an average incidence of approximately 8.6% (Nat. Acad. Sci., 1968; Young, 1968; Laster and Gregory, 1973; Young and Blair, 1974; Oxender and Adams, 1979; Patterson et al., 1979; Thornberry, 1979; Dennis, 1980; Khera, 1981). Of the PCM, 25% to 100% may be due to non-infectious causes such as dystocia, hypothermia, birth trauma, anoxia, acidosis, and congenital birth defects.

Non-infectious and unknown agents seem to be the major causes of reproductive failure. Nutrition is probably the most important environmental influence affecting reproduction (Cope, 1982). Non-infectious causes of abortion have been receiving increasing attention during the past decade (Miller, 1982; Stuart and Oehme, 1982). Generally,

the non-infectious causes can be grouped into five: nutritional, environmental stresses, toxins, genetic, and management.

Calf mortality due to dystocia is the largest single source of calf losses (Patterson et al., 1979). Laster and Gregory (1973) found that dystocic calves had a four times greater chance of mortality than those not experiencing dystocia. Dystocia is influenced by many factors such as birth weight, breed and sex of the calf, age and parity of the dam, breed of the sire, nutrition, and seasonal influences (Rice and Wiltbank, 1972; Laster and Gregory, 1973; Martin et al., 1975; Philipsson, 1976; Rice, 1979; Dennis, 1981).

Non-infectious factors are common causes of neonatal deaths. It is interesting to note that the gross lesions of calves dying from the so called "weak calf syndrome" are identical to those of hypothermia and exposure (Card et al., 1974; Dierks et al., 1976; Stauber, 1976; Olson et al., 1981). Uncomplicated starvation is an important cause of PCM (Young and Blair, 1974). Twin calves and calves from first calf heifers tend to victims of starvation more frequently than calves from multiparous cows. Lack of nourishment makes the calf more susceptible to chilling, hypothermia, and death from exposure.

Failure of passive transfer of maternal immunoglobulins (Ig) from poor or no colostrum ingestion is directly related to neonatal disease (Boyd, 1972; McGuire et al., 1976;

Dardillat et al;, 1978; Lumba et al., 1978; Davidson et al., 1981). There are seasonal variations in passive transfer of Ig to newborn calves (Gay et al., 1983) and hypothermia delays Ig absorption (Olson et al., 1980). Other reported causes of poor Ig transfer include restricted protein intake of the dam during the last trimester, dystocia, hormonal, and genetic (Blecha et al., 1981; Boyd and Hogg, 1981; Muggli et al., 1983).

Accurate identification of PCM problems within an operation is necessary before any reduction in incidence is possible. Improved management is essential for reducing calf losses and increasing reproductive efficiency (Patterson et al., 1979). Calf mortality in dairy cattle generally increases with herd size (Speicher and Hepp, 1973). This places much responsibility on management and management personnel. Management personnel was the only factor significantly related to the mortality rate in one study of PCM (Martin et al., 1975).

Reported here are the non-infectious causes of PCM from a three year study conducted in beef cattle herds in the Flint Hills of Kansas.

MATERIALS AND METHODS

The procedures used in this systematic necropsy study were described in Parts II and III. The general causes of death were divided into infectious, non-infectious, and

undetermined. Non-infectious factors determined were breed, sex and weight relative to time-of-death (TOD), cause of death, and type of congenital defects.

RESULTS

Non-infectious causes of PCM in the Kansas Flint Hills was found to be responsible for 59.4% of all calves examined from herds that agreed to submit all calf losses. This was significantly different when compared to non-contract calves ($P \leq .0001$). Non-infectious causes of death accounted for 108 of the 237 mortalities examined from both calf populations (45.6%). Operations with less than 150 cows tended to have a higher percent of non-infectious losses than those with more than 150 cows, 64.7% vs 52.9% ($P \leq .09$). Even though the incidence of non-infectious causes of PCM were different in commerical and purebred operations, 63.8% vs 54.7%, it was not significant. Annual influences during the 3 years appeared to be significantly related to the incidence of non-infectious causes of PCM, however, they were not statistically tested (50.0% vs 58.6% vs 65.9%).

Distribution of non-infectious PCM, from the contract calf population, into the 5 TOD categories was: 10.0% antepartum deaths, 66.7% early partum, and 97.7% late partum deaths, and 59.5% early postpartum, and 31.9% late postpartum deaths. The frequencies were significantly different ($P \leq .0001$). Fifty five percent of all non-infectious deaths occurred during the parturient period (46.7% late and 27.2% early).

Various non-infectious variables were recorded and analyzed so as to assess their influence on PCM and TOD. Parity: The parity of the dam was recorded in only 36.3% of the calves examined. Of those recorded, 28% of the losses came from primiparous heifers, 8% from cows in their second pregnancy, and 64% from multiparous cows. No statistical significance was from this data due to the lack of numbers. Breed: The breed was identified in all calves necropsied. The breeds and their respective incidence of PCM was: Angus 26.6%, Hereford 24.5%, Simmental 20.7%, Crossbreds 19.4%, Holstein 5.9%, and miscellaneous beef breeds 3.0%. The relationship between breeds and distribution of PCM within the 5 TOD categories is given in Table 1. Within the contract calf population, only beef breeds were examined. Of the calves dying from non-infectious causes (92), Herefords and Simmentals comprised the highest frequency (31.5% and 30.4%), followed by Angus, 22.8%. Crossbred calves were associated less frequently with non-infectious PCM than were purebred calves (15.2% vs 84.8%). The TOD distribution of the four breeds are listed in Table 2.

Sex: Sex of the calves necropsied was recorded in 232 of the 237 calves (97.9%). Sex was significantly related to mortality within both the early and late parturient categories ($P \leq .01$). Male calves accounted for 85.7% of the early partum and 65.3% of the late partum deaths compared to 14.3% and 34.7%, respectively, for females. There were no significant differences between sex and calf

mortality in the remaining 3 TOD categories. The overall incidence of male calves dying was significantly higher than females, 59.5% vs. 40.5% ($P \leq .01$).

Weight: Weights were recorded on 228 of the 237 calves (96.2%) necropsied. The mean calf weight regardless of TOD was 77 ± 30 lbs. with the lightest being 5 lbs. and the heaviest 275 lbs (Table 3). The overall weight of male calves was 72 ± 3 lbs. and females, 67 ± 5 lbs. There was no significant difference between the overall mean weights of the two sexes. Within the 5 TOD categories, there were no significant differences between the mean weights of the two sexes. Mean calf weight regardless of sex was significantly different ($P \leq .0001$) within the 5 TOD categories. Antepartum weights were significantly lower than either of the partum or postpartum categories ($P \leq .01$). The weight of calves dying late in parturition were significantly higher than those dying early after birth ($P = .03$). The mean, standard deviation and range of calf weights within the 5 functional TOD categories are summarized in Table 3.

Non-infectious causes

The majority of non-infectious causes of death in the calves necropsied could be allocated into 9 general conditions listed in decreasing order: dystocia, neonatal weakness, severe birth trauma, complicated starvation, starvation, natal nonviability, genetic defects, acute death, and abortion. Various general conditions and associated non-infectious causes are listed in Table 4.

Frequency of non-infectious causes of PCM identified included birth trauma 43.5%, anoxia/respiratory failure 15.7%, exposure 10.2%, uterine fluid aspiration 6.5%, starvation/mismothering 5.6%, genetic defects 5.6%, predation 1.9%, drowning 1.9%, exsanguination 1.9%, and miscellaneous 7.4%. Of the major cause, birth trauma, mild to moderate injury accounted for 76.6%, severe injury for 10.6% (hepatic rupture 60%, hemothorax 20%, and hemoperitoneum 20%), spinal column luxation 10.6%, and spinal fracture 2.1%. Of the spinal column luxation, 60% occurred between L1 and L2, 20% at T13 and L1, and 20% at the atlanto-occipital articulation. The fracture occurred at L1.

Congenital defects

Congenital defects were found in 8 of the 155 contract calves (5.2%); this was similar to the percent of congenital defects found in the total number of calves necropsied, 13 of 237 (5.5%). The defects were classified by the body system primarily involved (Table 5). Of all the congenital defects, visceral defects were the most frequent (46%), then skeletal (23%), central nervous system (15%), and multiple defects (15%). Fifty percent of the visceral defects involved the heart.

Congenital defects were found in all 5 of the functional TOD categories. Of the defects examined, 76.9% were associated with calves dying during the postparturient period (30.8% early and 46.1% late). Incidence of congenital defects did not vary significantly from year to year.

DISCUSSION

Non-infectious causes of PCM accounted for the majority of calf losses examined in this study. This was also found in previous studies (Laster and Gregory, 1973; Young and Blair, 1974; Patterson et al., 1979). The incidence was influenced primarily by the year (environmental influences) and size of the cow herd (management influences). The incidence of non-infectious types of PCM derived from randomly selected calves submitted for necropsy was not an accurate representation of the incidence occurring in the general calf population in the Kansas Flint Hills.

Within the 5 functional TOD categories, non-infectious PCM was most prevalent in the early partum, late partum, and early postpartum periods. Over a half of the deaths occurred some time during parturition. The three periods include the critical periods of parturition and neonatal adaptation. The majority of calf deaths are reported to occur during this time (Dennis, 1979, 1980). From this, it is concluded that the major factor in decreasing PCM is to decrease the non-infectious losses during birth and the first few days of life. Preventive efforts to decrease these losses should involve environmental protection and effective management policies. Research is needed to evaluate non-infectious preventive management practices and environmental stresses.

In this study, calf mortality did not increase with herd size as reported in dairy cattle (Speicher and Hepp, 1973). This only applies within the limits of the data

collected but many reasons may be theorized for the finding. The most obvious being the primary objective of a beef herd is to deliver viable marketable calves in contrast to milk production in a dairy herd. Management practices within the two different cattle operations are then targeted to maximize their primary objective.

The influence of parity of dam on PCM was unable to be sufficiently examined in this study. Previous work suggested that parity significantly influenced PCM as the incidence of dystocia was 2 to 4 times higher in primiparous as compared to multiparous cows (Rice and Wiltbank, 1972; Oxender et al., 1973; Philipsson, 1976).

Purebred calves were found to be victims of non-infectious PCM more frequently than crossbred calves. This agreed with previous reports indicating that crossbred calves could tolerate higher levels of stress associated with difficult birth better than purebreds and that the higher mortality rate in calves experiencing dystocia were related to calf stress resulting from delayed and difficult parturition (Laster and Gregory, 1973).

Of the purebred calves necropsied, the association with the overall incidence of non-infectious PCM was relatively equal, but the distribution by TOD varied. Angus calves were associated less with late parturient deaths and Hereford calves less so with late postpartum deaths.

The effect of sex on PCM during the parturient period was significantly higher ($P \leq .01$) in male than female calves. This agreed with previous reports on the influence of sex on calf mortality during parturition (Laster and Gregory, 1973). Birth weight was not significantly different between the two sexes of calves during this period indicating that the sex difference in mortality was not due to differences in birth weight. Laster and Gregory (1973) found that birth weight has a significant effect on parturient calf mortality. Since there was no difference between the birth weight of the two sexes, it can be postulated that the reason for the sex influence on parturient calf mortality is probably the female calf's ability to better withstand the stresses of parturition and adaptation.

There were extreme variation in the weight of the dead calves ranging from 5 lbs. to 275 lbs. This agrees somewhat with a previous report that a relatively higher percentage of extremely light and heavy calves are born dead (Rice, 1979).

The major non-infectious causes of PCM in this study were related primarily to dystocia. Gross signs indicating the degree of dystocia were used to differentiate most of these calves; anoxia, mild to moderate birth trauma, and severe identifiable birth trauma were manifestations of the increasing degrees of dystocia. Weak calves dying after birth from exposure could often be linked to the stress of a difficult birth. The major influence of birth trauma on PCM

was confirmed and reported by Gopal (1977) in a study also conducted in the Flint Hills area. Many lesions of severe birth trauma observed by Gopal were also identified in this study.

The incidence of congenital defects found in this study (0.42%) was within the reported range of 0.2% to 3% (Laster and Gregory, 1973; Leipold, 1978, 1980; Leipold et al., 1983). Congenital defects were found in all of the 5 functional TOD categories. The defects examined were not as varied as those reported previously in this area by Gopal (1977); however, the order and frequency of visceral, skeletal and CNS defects were relatively the same. Various cardiac defects accounted for 23% of all congenital defects examined. Calves dying of cardiac defects died early in the postpartum period as they were unable to effectively adapt to the neonatal circulatory pathway. Internal hydrocephalus was the most common CNS defect and these calves died in the late postpartum period.

SUMMARY

A 3-year study into the causes of perinatal calf mortality (PCM) of beef herds in the Flint Hills of Kansas is reported. Systematic necropsy was performed on 237 calves and all were classified by their time-of-death (TOD). Incidence of non-infectious causes of PCM was 59.4%. Non-infectious causes were determined in 108 calves (45.6%). Non-infectious incidence of PCM was influenced by year

(environmental factors) and size of the cow herd (management factors) ($P \leq .09$). Non-infectious causes were most prevalent during the early partum, late partum, and early postpartum periods with 55% of all non-infectious PCM occurring during parturition. Crossbred calves were less associated with non-infectious perinatal mortality than purebred calves (15.2% vs 84.8%). Male calves were associated with a higher ($P \leq .01$) incidence of PCM during the early and late parturient categories compared with females (85.7% and 65.3%, respectively, for males vs. 14.3% and 34.7%, respectively, for females). There was no significant difference between weights of the two sexes. Mean calf weight was 77 ± 30 lbs. with a wide range (5 to 275 lbs.). Dystocia and severe birth trauma in combination accounted for 51.9% of all of the non-infectious causes. Most prevalent non-infectious causes were birth trauma 43.5%, anoxia/respiratory failure 15.7%, and exposure 10.2%. Congenital defects were observed in 5.2% of the contract calves examined. Visceral defects (46%) were the most prevalent followed by skeletal (23%), CNS (15%), and multiple defects (15%). Various cardiac defects accounted for one half of the visceral defects, and internal hydrocephalus was the only CNS defect.

REFERENCES

Blecha F, Bull RC, Olson DP, Ross RH, Curtis S. Effects of prepartum protein restriction in the beef cow on immunoglobulin content in blood and colostral whey and subsequent immunoglobin absorption by the neonatal calf. J Anim Sci 1981;53:1174-1180.

Boyd JW. The relationship between serum immune globulin deficiency and disease in calves: a farm survey. Vet Rec 1972;90:645-649.

Boyd JW, Hogg RA. Field investigations on colostrum composition and serum thyroxine, cortisol and immunoglobulins in naturally suckled dairy calves. J Comp Path 1981;91:193.

Card CS, Spencer GR, Stauber EH, Frank FW, Hall RF, Ward ACS. The weak calf syndrome -- epidemiology, pathology and microorganisms removed. Proc 77th Ann Mtg US Anim Hlth Assoc 1974;77:67-72.

Cope GE. Estrus synchronization and reproduction management of beef herds in the south. Proc 14th Ann Conv Am Ass Bov Pract 1982;No 14:104-107.

Dardillat J, Trillat G, Larvor P. Colostrum immunoglobulin concentration in cows: relationship with their calf mortality and with the colostrum quality of their female offspring. Ann Rech Vet 1978;9:375-384.

Davidson JN, Yancy SP, Campbell SG, Warner RG. Relationship between serum immunoglobulin values and incidence of respiratory disease in calves. JAVMA 1981;179:708-710.

Dennis SM. Perinatal mortality of ruminants. Compend Cont Ed Pract Vet 1979;1:S17-S26.

Dennis SM. Investigating perinatal calf mortality. Proc Ann Mtg Soc Theriogenol. Omaha NE, September 10-12, 1980, pp 150-168.

Dennis SM. Low viability of calves at birth. Vet Ann 1981;21:63-73.

Dierks RE, Smith MH, Gollehon D. Isolation and characterization of adenoviruses from aborted fetuses and calves with weak calf syndrome. Proc 19th Ann Mtg Am Assoc Vet Lab Diag 1976;395-404.

Gay CC, McGuire TC, Parish SM. Seasonal variation in passive transfer of immunoglobulin G1 to newborn calves. JAVMA 1983;183:566-568.

Gopal T. Investigations into prenatal and perinatal mortalities among calves. PhD thesis, Kansas State University, 1977.

Khera SS. Fetal and young calf mortality among bovine farmstock in India. Indian J Anim Sci 1981;51:292-302, 425-431, 432-438.

Laster DB, Gregory KE. Factors influencing peri- and early postnatal calf mortality. J Anim Sci 1973; 37:1092-1097.

Leipold HW. Genetics and disease in cattle. Proc 11th Ann Conv Am Assoc Bovine Pract, Baltimore, MD. December 1978;11-14, 18-31.

Leipold HW. Diagnosis and control of undesirable genetic diseases and lethal factors in cattle. Proc XI Intern Congr Dis Cattle, Spain. 1980;543-555.

Leipold HW, Huston K, Dennis SM. Bovine congenital defects. Adv Vet Sc Comp Med 1983;27:198-272.

Lumba F, Fumiere I, Tshibangn M, Chauvaux G, Bienfet V. Immunoglobulin transfer to calves and health problems in large bovine units. Ann Rech Vet 1978;9:353-360.

Martin SW, Schwabe CW, Franti CE. Dairy calf mortality rate: influence of meterologic factors on calf mortality rate in Tulare County, California. Am J Vet Res 1975; 36:1105-1109.

McGuire TC, Pfeiffer NE, Weikel JM, Bartsch RC. Failure of colostral immunoglobulin transfer in calves dying from infectious disease. JAVMA 1976;169:713-718.

Miller RB. A discussion on reproductive failure in cattle. Bovine Pract 1982;17:39-51.

Muggli NE, Hohenboken WD, Cundiff LV, Kelley KW. Inheritance of maternal IgG_1 concentration by the bovine neonate. J Anim Sci 1983;57(Suppl. 1):162.

National Academy Science Subcommittee on Prenatal and Postnatal Mortality in Cattle, 1968; Publ no 1685, Washington, DC. pp 1-130.

Olson DP, Bull RC, Kelley KW, Ritter RC, Woodard LF, Everson DD. Effects of maternal nutrition restriction and cold stress on young calves: clinical condition, behavioral reactions, and lesions. Am J Vet Res 1981;42:758-762.

Olson DP, Papasian CJ, Ritter RC. The effects of cold stress on neonatal calves. 2. Absorption of colostral immunoglobulins. Can J Comp Med 1980;44:19-23.

Oxender W, Adams W. Problems associated with calving and neonatal period in beef cattle. In: Mason et al., ed. Calving Problems and Early Viability of the Calf. Commission of the European Communities, Martin Nijhoff Publishers, The Hague, 1979;408-422.

Oxender WD, Newman LE, Morrow DA. Factors influencing dairy calf mortality in Michigan. JAVMA 1973;162:458-460.

Patterson DJ, Bellows RA, Burfening PJ, Short RE, Carr JB. Incidence and causes of neonatal and postnatal mortality in range cattle; USDA, SEA and Montana State University. J Anim Sci 1979;49(Suppl. 1):325.

Philipsson J. Calving performance and calf mortality. Livest Prod Sci 1976;3:319-331.

Rice LE. Perinatal management of calves. Proc Ann Mtg Soc Theriogenol, Mobile, Alabama, 1979;138-150.

Rice LE, Wiltbank JN. Factors affecting dystocia in beef cattle. JAVMA 1972;161:1348-1358.

Speicher JA, Hepp RE. Factors associated with calf mortality in Michigan dairy herds. JAVMA 1973;162:463-466.

Stauber EH. Weak calf syndrome: a continuing enigma. JAVMA 1976;168:223-225.

Stuart LD, Oehme FW. Environmental factors in bovine and porcine abortion. Vet Human Tox 1982;24:435-441.

Thornberry H. Conclusions from the EEC Seminar on perinatal ill-health in calves. In: Mason et al., ed. Calving Problems and Early Viability of the Calf, Commission of the European Communities, Martin Nijhoff Publishers, The Hague. 1979;487-493.

Young JS. Breeding patterns in commercial beef herds. Aust Vet J 1968;44:350-356.

Young JS, Blair JM. Perinatal calf losses in a beef herd. Aust Vet J 1974;50:338-344.

TABLE 1 - Breed of calf relative to time-of-death

	APD		EPD		LPD		EPPD		LPPD		Total	
Breed	N	%	N	%	N	%	N	%	N	%	N	%
Angus	5	7.9	5	7.9	8	12.7	22	34.9	23	36.5	63	26.6
Hereford	9	15.5	3	5.2	18	31.0	19	32.8	9	15.5	58	24.5
Simmental	5	10.2	3	6.1	16	32.7	10	20.4	15	30.6	49	20.7
Crossbred	7	15.2	2	4.4	6	13.0	3	6.5	28	60.9	46	19.4
Misc. Beef breed	1	14.3	1	14.3	0	0	0	0	5	71.4	7	3.0
Holstein	3	21.4	0	0	1	7.1	0	0	10	71.4	14	5.9
Total	30	12.7	14	5.9	49	20.7	54	22.8	90	38.0	237	100

APD = antepartum death
EPD = early partum death
LPD = late partum death
EPPD = early postpartum death
LPPD = late postpartum death

TABLE 2 - Breed of calf dying of non-infectious causes relative to time-of-death

Breed	Antepartum		Early Partum		Late Partum		Early Postpartum		Late Postpartum		Total	
	%	N	%	N	%	N	%	N	%	N	%	N
Angus	0.0	(0)	9.5	(2)	28.6	(6)	38.1	(8)	23.8	(5)	22.8	(21)
Hereford	3.5	(1)	6.9	(2)	58.6	(17)	27.6	(8)	3.5	(1)	31.5	(29)
Simmental	0.0	(0)	7.1	(2)	50.0	(14)	25.0	(7)	17.9	(5)	30.4	(28)
Crossbred	0.0	(0)	14.3	(2)	42.9	(6)	14.3	(2)	28.6	(4)	15.2	(14)
Total	1.1	(1)	8.7	(8)	46.7	(43)	27.2	(25)	16.3	(15)	100	(92)

TABLE 3 - Weight of calves dying during the perinatal period

TOD Category	N	Mean (lbs)	S.D.(lbs)	Range (lbs)
Antepartum	25	46[abcd]	21	5-90
Early Partum	14	73[a]	13	55-100
Late Partum	47	90[be]	18	57-131
Early Postpartum	54	73[ce]	21	18-115
Late Postpartum	88	84[d]	36	31-275
Totals	228	77	30	5-275

[abcd]Mean weights significantly different ($P \leq .01$)

[e]Mean weights significantly different ($P = .03$)

TABLE 4 - Cause of non-infectious conditions associated with perinatal calf mortality

Condition	N	Cause	N
Dystocia	45	Birth trauma (mild to moderate)	31
		Anoxia	8
		Uterine fluid aspiration	5
		Natal nonviability	1
Neonatal Weakness	22	Exposure	6
		Birth trauma (mild to moderate)	5
		Respiratory failure	4
		Uterine fluid aspiration	2
		Drowning	1
		Heart failure	1
		Predation	1
		Premature birth	1
		Exanguination	1
Severe Birth Trauma	11	Spinal column luxation	5
		Hepatic rupture	3
		Hemothorax	1
		Hemoperitoneum	1
		Spinal column fracture	1
Complicated Starvation	7	Exposure	5
		Incarcerated bowel	1
		Predation	1
Starvation	6	Mismothering	6
Natal Nonviability	6	Anoxia	5
		Exanguination	1
Genetic Defects	6	Skeletal	3
		Visceral	2
		CNS	1
Acute Death	3	Drowning	1
		Acidosis	1
		Pulmonary & cerebral edema	1
Abortion	2	Environmental	2
Total	108		108

TABLE 5 - Congenital defects relative to time-of-death

Defect	Antepartum	Early Partum	Late Partum	Early Postpartum	Late Postpartum	Total
I. Visceral						6
Low septal defect				1		
Tetralogy of Fallot				1		
Patent ductus arteriosus				1		
Atresia ilei					1	
Pyloric stenosis					1	
Congenital goiter	1					
II. Skeletal						3
Brachycephaly		1				
Campylognathia			1			
Torticollis				1		
III. Central nervous system						2
Hydrocephalus					2	
IV. Multiple systems						2
Hydrocephalus and patent ductus arteriosus					2	
Total	1	1	1	4	6	13

V. NECROPSY PROCEDURES FOR INVESTIGATING PERINATAL CALF MORTALITY

INTRODUCTION

Significant reduction and constant monitoring changes in perinatal calf mortality (PCM) are two primary goals in an effective cow-calf health and production management program. New insights into the relative importance of factors influencing calf mortality have been provided by necropsy findings correlated with fetal, natal and neonatal problems and their corresponding time-of-death (TOD) (Dennis, 1979). A necropsy method for investigating perinatal lamb losses has been reported (McFarlane, 1965). Specific TOD categories were suggested as an effective means of investigating PCM (Young and Blair, 1974; Dennis, 1979, 1980, 1981). This concept and technique is useful for identifying, defining and monitoring problems and changes in PCM in individual herds and for entire areas.

The purpose of this paper is to describe the necropsy method used to investigate PCM in the Flint Hills area of Kansas.

NECROPSY

Each calf was identified by number. A short history was taken and each calf was weighed to the nearest pound. Necropsy began with the calf lying on its left side, the ventral abdomen and feet towards the prosector. A careful external examination was done to determine the presence of

meconium staining (Fig 1), generalized or localized edema of the head (Fig 2), neck, limbs and perineum, gross signs of birth trauma, gross developmental defects, degree of hydration, postmortem autolysis, coat condition, condition of the umbilicus, and the hooves examined to see if the calf had walked or not (Fig 3 and 4). Length of hair and development was assessed and if preterm, gestational age was estimated (Table 1).

The right front limb was lifted and the skin and muscles attaching it to the sternum and thorax were cut. The leg and the attached skin was reflected dorsally. Blood samples were then taken from the axillary blood vessels by compressing the thorax and drawing the blood into a 12 cc plastic syringe. The blood was transfered to a 10 cc glass tube for later use. The upper rear leg is was then grasped and an incision is made through the adductor, gracillis and quadricepus muscles, the coxofemoral joint was disarticulated and the limb reflected dorsally. The sagittal incision was continued from the front leg posteriorly to the anus and anteriorly to the symphysis of the mandible. The presence and thickness of any subcutaneous edema in these areas was noted (Fig 2 and 5). The skin over the right side of the mandibular, cervical and abdominal regions was then reflected dorsally.

The upper jaw was removed by inserting the knife lateral to the ramus and under the masseter muscles and severing them from the lateral surface of the angle of the rami. The

mandibular symphysis was split with bone rongeurs, saw or rib shears. The upper ramus was grasped and pulled back cutting the muscles medial to the ramus going around the posterior border of the angle of the ramus. The freed ramus was then pulled posteriorly, rotated laterally, disarticulated and removed by cutting any remaining muscular attachments. The oral cavity and surface of the tongue was examined for any erosions, ulcers, gingival hyperplasia and other lesions. The tongue was severed from the medial surface of the down rami. The cut was extended behind the hard palate and around the pharynx to the level of the hyoid bones that were disarticulated. The tongue, pharynx, esophagus and trachea were recovered to the thoracic inlet. Special notice should be made of the size and appearance of the thymus that normally extends anteriorly from the thoracic cavity up to one-third to one-half of the neck. The esophagus and trachea were then opened and examined. Any hyperemia, fluid, hemorrhage, and erosions in the trachea and esophagus were noted.

The abdomen was opened by cutting the muscles along the costal arch dorsally to the lumbar muscles, posteriorly to the wing of the ileum, ventrally to the midline, and the lateral abdominal wall was reflected ventrally. Abdominal organs were then examined <u>in situ</u> (Fig 6). Presence of any abdominal fluid was determined and samples were collected for examination and culture. Amount, character, color, and presence of fibrin in abdominal fluid was observed. The rib

cage was then removed by using rib shears or a knife inserted into the thorax just caudal to the xiphoid cartilage and cutting cranially through the soft sternal cartilage to the thoracic inlet. The dorsal rib attachments were either cut with rib shears starting at the level of the transverse process of the first lumbar vertebra cranially to the dorsal aspect of the first rib; more often the ribs were easily reflected dorsally and disarticulated by hand. The rib cage was then freed of its attachment to the diaphragm. At this time, the thoracic organs and fluid were examined in situ and any specimens for culture taken (Fig 7). Pericardial, thoracic or abdominal fluids for darkfield examination were also collected at this time. Thoracic and pericardial fluid should be examined for quantity, character, color, and presence of fibrin.

At this stage the internal organs were removed and systematically examined. The tongue, esophagus and trachea were grasped and with dorsal-caudal traction, the thoracic viscera were reflected and removed from the thoracic cavity. The rib cage and thoracic vertebrae were examined for evidence of birth trauma and fibrin tags. The lungs were palpated and degree of aeration estimated and pathology is noted. The tracheal incision was extended into the bronchi and bronchioles with scissors and the presence of fluid and meconium was noted (Fig 8). The heart was held in the left hand with the right ventricle on left and the apex pointing upwards. It is examined for degree of pericardial fat

metabolism and hemorrhages. The knife is inserted at the apex of the right ventricle through the pulmonary valve into the pulmonary artery. The ductus arteriosus is then easily examined on the greater curvature of the pulmonary artery (Fig 9). A similar procedure was followed on the left side of the heart and when both sides were opened a finger could be passed from right to left atrium and the patency of the formen ovale noted (Fig 10).

The liver, spleen, stomachs and intestines were removed by severing their attachments at the root of the mesentery and diaphragm, and cutting through the terminal colon. The presence or absence of meconium and feces and their nature and consistency were noted. The spinal column and abdominal wall were then examined. The abdominal organs were systematically examined starting with the liver and checking for evidence of infection, congestion, or rupture. The abomasum was opened and the amount and character of its contents were evaluated. The abomasal mucosa was examined for any erosions. The small and large intestines were examined for defects, hyperemia, hemorrhage, and inflammation and the nature of the contents evaluated. The mesentery was examined evidence of inflammation and absorbed milk in the lacteals. The mesenteric lymph nodes were examined for size, consistency and hemorrhage.

The umbilicus was examined for size, moistness, dehydration, hyperemia, edema, hemorrhage and inflammation. Special attention was paid to the immediate abdominal wall

surrounding the umbilicus, both external and internal. The ruptured ends of the umbilical arteries were carefully with a small pair of pointed scissors for the presence and absence of clots (Fig 11). When present, the consistency of the clots, soft or firm, was noted. The patency of the urachus was evaluated. The metabolic state of the perirenal fat was observed and assessed (Fig 12 and 13). The kidneys were then removed, incised transversely, and the degree of any autolysis was assessed (Fig 14 and 15). The bladder was incised and the amount and nature of urine was evaluated.

Initially each necropsy was complete with the cranial cavity being opened and meninges and brain being examined together with the endocrine system. With experience and absence of obvious gross lesions in the CNS, oral cavity and endocrine organs; these organs were examined superficially. The necropsy method was rapid and many calves could be examined by this systematic approach at any time -- 10 calves per hour by one operator.

In this study samples for diagnostic testing, histopathology and time-of-death classification were all performed along with the standardized necropsy. All information was recorded on a special perinatal calf necropsy examination form (Appendix 4). A summary of gross necropsy findings and a tentative diagnosis were recorded on the standard necropsy form in triplicate and copies were sent to the consulting veterinarian and the diagnostic laboratory (Appendix 3). At the end of each year, a summary of calf mortality data was sent to each owner (Appendix 8).

TIME-OF-DEATH CLASSIFICATION

Time-of-death classification (TOD) was made during necropsy by identifying and understanding the importance of presence or lack of certain gross findings. To effectively do this, two concepts must be understood:

1. Physiological sequence of events a calf goes through before, during and after birth and their effect on the calf's viability..
2. Postmortem changes in various body tissues.

1. Physiological Events of Parturition and Adaptation

Perinatal physiology and adaptation was reviewed by Randall (1978). The bovine uterus is essentially ready for parturition from approximately day 200 of gestation till term. Initiation of parturition is primarily a hormonal process and the fetus determines primarily the length of gestation and dominates the mechanisms initiating it.

How the fetus determines when to initiate parturition is poorly understood, but some believe that the degree of fetal stress is the cause. Whatever the cause, it stimulates the fetal hypothalamus to release corticotropin releasing factor which acts on the pituitary gland causing release of adrenocorticotropin hormone, which in turn, causes the fetal adrenals to release steroids that stimulate fetal prostaglandin release, the fetal pituitary-adrenal axis. Any hypo- or hyperactivity of the axis may lead to delayed or premature birth.

Fetal prostaglandins along with placental prostaglandins (stimulated from rising fetal and maternal estrogen levels) effect the dam at three important sites: 1) pituitary gland, stimulating oxytocin release; 2) ovaries, producing a luteolytic effect and relaxin release; and 3) uterus, which along with oxytocin and estrogen, increase myometrial activity. Prostaglandins directly effect the placenta by inhibiting progresterone production and releasing relaxin. The outcome of this sequence of events is dilation of the cervix, increased myometrial activity, and subsequent expulsion of the fetus.

Stage two of parturition (passage of the fetus through the birth canal) in a cow lasts 30 minutes to 4 hours. The calf is at a high level of metabolic activity at this time and uses up much energy during parturition. In passage through the birth canal, the umbilical cord is often occluded for periods of time producing fetal anoxia. Normal fetuses can easily tolerate short periods of anoxia. The high level of fetal activity and prolonged anoxia can result in metabolic acidosis and the fetus responds by gasping in utero. Results of fetal anoxia can vary from no significant effect to fetal asphyxiation.

Along with mechanical forces causing fetal anoxia, considerable pressure is exerted on the fetus as it passes through the birth canal. Even though harmful at times, this pressure is important in preparing the fetus for its first breath.

Rupture of the umbilical cord signifies the beginning of the neonatal adaptation period. Adaptation normally occurs in the following sequence: respiration, circulation, thermoregulation, mobility, nutrition and microbial defense. To advance in this sequence the calf must effectively adapt to each physiological change preceding it.

The key mechanisms to each step in the normal calf are as follows: In respiration the stimulation of the respiratory centers by a build up of CO_2 during parturition as well as the elastic recoil of the thorax after its compression through the birth canal (Head's paradoxial reflex) are the most important factors in expansion of the lungs and initiation of the first breath. Then the calf must convert its fetal cardiovascular pathway to the adult pathway. For this to happen the umbilical arteries must be sealed, the foramen ovale must close, and the ductus arteriosus must constrict. In a neonate with a functional heart, the elastic umbilical arteries recoil when the umbilicus is severed and seal off at the ruptured ends and the trapped blood clots.

When the placental blood pressure is lost, the pressure in the right side of the heart becomes less than the pressure exerted by the left side, causing an increased left atrial pressure and mechanical closure of the foramen ovale. Last to functionally close is the ductus arteriosus which is stimulated to close by the increased oxygen tension of the blood. Complete closure usually takes a few weeks.

The neonate must then adapt to the environmental temperature that is important for survival and continued adaptation. The calf can usually stand within 30 minutes after birth and is walking soon after. If it takes a calf longer than one hour to stand, it should be considered potentially abnormal. After the calf stands, the next adaptive hurdle it must overcome is that of obtaining food. This step is dependent on a cooperative dam as well as the complex sucking and affinity reflex (thigmotaxis) that occurs between the calf and its dam.

By ingesting colostrum, the calf had overcome the last two steps in adaptation; nutrition and acquiring maternal antibodies. This passive immunity is important because the newborn calf is not functionally immunocompetent. From the stress of birth and adaptation, the calf has high levels of circulating corticosteriods that inhibit its inherent defenses thereby compounding its vulnerability from agammaglobulinemia by lymphopenia and decreased phagocytic activity. The calf must ingest the maternal antibodies within the first 24 hours after birth for absorption; maximum absorption occurs during the first 6 to 8 hours.

Now that the calf has effectively completed the adaptive process, it need only to maintain them to survive.

2. Postmortem Changes

In general, the two biggest influences on postmortem changes is the death-to-necropsy time interval and the

ambient temperature (Dennis, 1980). The death-to-necropsy time interval can usually be controlled or can be determined from the history. The temperature and humidity in utero is high and during parturition, the calf's activity level increases its temperature. After parturition, the temperature surrounding a calf's environmental temperature is usually markedly decreased and the calf may have difficulty in maintaining its body temperature.

Postmortem changes characteristic of antepartum death depend on how long the fetus was dead in utero before expulsion. Fetal age is estimated by its state of development (Table 1). All antepartum deaths lack signs of viability and survivability, ie. umbilical artery clot, localized edema, and lung aeration. Therefore, the only thing you have to assess is the degree of tissue autolysis and hydration.

Due to the higher intrauterine temperature, a fetus dying in utero will have rapid autolysis of the renal cortex, starting within 30 minutes after death (Fig 15). At this time the liver will also show a degree of softening. In contrast, renal cortical autolysis in postpartum deaths is usually not evident for 24 to 48 hours. The next prominent intrauterine autolytic change is evident from approximately 12 hours after death, hemolysis and hemoglobin imbibition. Generalized hemoglobin staining of all body tissues is complete by 30 to 36 hours after death in utero (Fig 6 and 16).

Fetuses dying greater than 48 hours before expulsion will have advanced autolysis and varying degrees of generalized edema. Edematous fetuses may be aborted between 4 to 8 months of gestation. Other gross changes in fetuses dying *in utero* between 3 and 8 months of gestation may be maceration, mummification, or emphysema. For fetal emphysema to occur, the cervix has to be patent and putrefactive bacteria to invade from the vagina. If death occurs before 90 days of gestation and the fetus is not aborted, complete resorption is possible as fetal bones have not yet ossified.

Neonates dying during parturition can be mistaken as abortion deaths and are often presented as such. Both deaths occur *in utero*, therefore, the gross signs may be similar. Autolysis is the most common finding and edema the second. The key in differentiating a parturient from an antepartum death is that a fetus dying during parturition may have gross signs of viability during parturition and absence of signs indicating survival after parturition.

Localized edema is the most important finding indicating viability during birth. The degree of localized edema indicates the time the calf survived the birth process with a functional heart. The edematous fluid accumulates in organs protruding from the vagina during a prolonged or difficult birth due to localized venous constriction. Common sites for localized edema are the head, tongue, submandibular space, cervical area, fore or hindlegs, and

rear quarters (Fig 2 and 5). In contrast, the degree of in utero renal cortical autolysis indicates the time the calf was dead during the birth.

Another important finding indicating viability during parturition is meconium staining of the haircoat due to fetal stress from hypoxia in utero (Fig 1). If the hypoxia persists, the fetus attempts respiration that may result in inhalation of uterine fluids and meconium and possibly pneumonia if it survives birth (Fig 8). The hypoxia also produces petechial hemorrhages on the pleura, epicardium, endocardium and thymus.

Frequently, lungs of a calf dying during parturition may be partially expanded and these deaths are often confused with those dying immediately after birth. Calves dying during parturition, however, lack the most important and first sign of survivability, a clot in the umbilical arteries.

Gross findings characteristic of postpartum death are those indicating survival of the birth process and successful or unsuccessful steps in the adaptation sequence of events. All postpartum deaths can be identified by a clot in the umbilical arteries indicating that the calf survived birth with a functional heart (Fig 11). Gross evidence of fetal stress (meconium staining) and severity of birth (localized edema and hemorrhages) may also be observed. After survival of the birth has been determined you need only to identify at which stage of the adaptation process

the calf died. The lungs are examined and the degree of aeration estimated (Fig 7). This indicates how effectively the calf converted from hematogenous to aerogenous respiration. The size of the opening of the ductus arteriosus is important; if greater than 6 mm (thickness of a pencil) in a calf 5 to 7 days old or older it should be considered significant. A decrease in oxygen tension in the blood (hypoxia) may stimulate the ductus to reopen, returning the calf to a form of fetal circulation and often death. This may complicate cases of pneumonia during the first two weeks of life.

The presence or absence of the eponychium (horny tissue covering the bottom of the fetal hoof) indicates when the calf walked (Fig 3 and 4). Presence of milk in the abomasum, confirms whether the calf had suckled. Examination of the lacteals of the small intestinal mesentery indicate whether ingested milk is being absorbed. Presence or absence of meconium should be noted as normally it is voided during the first 24 hours after the calf has suckled. Finally, the amount of visible fat present on the pericardium, around the kidneys, and between the ribs adjacent to the sternum (primarily brown fat) should be assessed and the degree of metabolism noted as nil, moderate or complete (Fig 12 and 13).

3. Classifying Perinatal Calf Mortality

Time-of-Death

The five functional categories of TOD (McFarlane, 1965) based on gross findings are:

1. Antepartum deaths
2. Early parturient deaths
3. Late parturient deaths
4. Early postpartum deaths (24 to 48 hours postpartum)
5. Late postpartum deaths (3 to 28 days)

The gross characteristics of the five categories are:

1. Antepartum Deaths: Characterized by fetal death and no signs of viability. Major gross findings include no clots in the umbilical arteries, renal cortical autolysis, generalized edema that may be hemoglobin stained, and hemoglobin-stained tissues.

2. Early Parturient Deaths: Characterized by fetal death with weak and often subtle signs of viability. Major gross findings include no umbilical artery clots; no hemoglobin-stained tissues; and the lack of localized or generalized subcutaneous edema. Other findings may include moderate, marked, or no renal cortical autolysis; meconium staining of the carcass; partial lung aeration; and petechial hemorrhages on the thymus, pleura, pericardium, and epicardium.

3. Late Parturient Deaths: Characterized by fetal death but with distinctively positive signs of viability.

Major gross findings include no thrombus in umbilical arteries, and the presence of localized subcutaneous edema of the head, limbs or perineum.

4. Early Postpartum Deaths: Characterized by signs of being liveborn but neonatal adaptation unsuccessful and immediate death. Major gross findings include a soft to firm thrombus in the umbilical arteries, and slight to no body fat metabolism. Other findings may include partial, complete, or no lung aeration; may or may not have walked; may or may not have fed; and no intestinal absorption of milk.

5. Late Postpartum Deaths: Characterized by signs of being liveborn, unsuccessful (or more commonly) successful neonatal adaptation but failure to sustain life resulting in a delayed death. Major gross findings include a thrombus in the umbilical arteries and moderate to complete body fat metabolism. Other findings often seen include all of the above in the early postpartum deaths except that usually these calves have nursed, with food having passed completely through the digestive tract.

IV. PROBLEM-ORIENTED APPROACH TO DIAGNOSIS:

This approach was designed to compare various parts of the final diagnosis with TOD and other variables. It consisted of assessing the general cause or nature of the PCM as being infectious, non-infectious or undetermined and then

developing a three part diagnosis comprised of (1) a problem title or general condition, (2) gross diagnosis(es), (3) and specific cause(s).

The general cause and problem title were initially derived from information gained from the history and gross findings. They were then updated and refined, if possible, by adding the gross diagnosis(es) and specific cause(s) as determined by further examinations and diagnostic testing.

Ten general problem titles were utilized to define the general condition: abortion, acute death, birth trauma, neurologic dysfunction, disease complicated starvation, congenital defects, dystocia, fetal weakness, neonatal weakness, and uncomplicated starvation. Gross diagnoses were many but standardization was employed; examples include enteritis, septicemia, meningitis, heart defect, skeletal defect, etc. Specific causes included infectious agents, non-infectious conditions, and specific defects, for example: E. coli, rotavirus, exposure, patent ductus arteriosus, hydrocephalus, etc.

All three parts of the final diagnosis may not be defined. This is the case many times and often the specificity of the diagnosis depends on the extent of the diagnostic workup. The general cause and problem title can almost always be determined. Examples of completed final diagnoses included: infectious/complicated starvation/enteritis/rotavirus; undetermined/neonatal weakness/; non-infectious/abortion//heat stress; and so on.

V. SUMMARY

The necropsy procedure proved to be repeatable and fairly objective. Most ranch managers and owners have the ability to classify calf deaths by TOD and this may be the key in being able to use this technique in monitoring PCM in a large number of herds. The TOD can always be determined even from a relatively autolytic carcass. Whereas, frequently the actual cause of death is left unknown after most routine necropsies. An experianced veterinarian is left then with the task of deciding which, if any, diagnostic tests should be used. It is these cases where the TOD classification is most beneficial because you have a way of comparing that calf with others in the same classification and obtaining a list of differential diagnoses and probabilities for that particular herd or region. Then you can objectively decide what further diagnostic tests may be needed.

With this information and the aid of a computer for storage, retrieval, manipulation and comparison of data, you can accurately assess the incidence and distribution of PCM and identify causative factors involved in a client's problem. Then you can offer management/preventive steps based on economics and individual needs of the client.

When this is done for a group of clients in a similar geographic or environmental region, all data in that region can be combined and an area incidence and distribution of PCM can be determined as well as the frequency of the

various categories of the final diagnosis. This can then serve as base data or regional "normals" from which to compare all herds in that geographical area. Comparisons should be made yearly to identify new problems but more importantly, they can be made between years or over a longer time so as to visualize disease trends and effectiveness of management practices.

Cow-calf health and production management systems need to be adjusted to fit the specific conditions and resources that vary between operations and regions. Applying the proposed necropsy procedure to the problem of PCM will give information for individually designing herd health/ production management programs to monitor changes and increase the profitability of your client's beef cow operation.

REFERENCES

Dennis SM. Perinatal mortality of ruminants. Compend Cont Ed 1979;1:S17-S26.

Dennis SM. Investigating perinatal calf mortality. Proc Ann Mtg Society Theriogenol Omaha, NE, Sept 10-12, 1980, pp 150-168.

Dennis SM. Low viability of calves at birth. Vet Annual 1981;21:63-73.

Hubbert WT, et al. Recommendations for standardizing bovine reproductive terms. Cornell Vet 1972;62:216-237.

McFarlane D. Perinatal lamb losses: 1. An autopsy method for the investigation of perinatal losses. N Z Vet J 1965;13:116-135.

Randall GCB. Perinatal mortality: Some problems of adaption at birth. Adv Vet Sci Comp Med 1978;22:53-81.

Roberts SJ. Veterinary Obstetrics and Genital Diseases, 2nd ed., Publ by Author, Ithaca, NY, 1971;447-472.

Young JS, Blair JM. Perinatal calf losses in a beef herd. Aust Vet J 1974;50:338-344.

TABLE 1 - Gestational age and fetal characteristics*

Days of gestation	Body weight	Crown-Rump Length (cm)	External Appearance
60	8-30g (.25-1oz)	6-8 (2.5-3.25")	Claw buds and small scrotum recognizable, eyelids cover eyes
90	200-400g (6-13oz)	13-17 (5-6.5")	Hooves firm, hair on lips, chin and eyelids, scrotum present
120	0.8-2.0kg (2-4 lbs)	22-32 (8.5-12.5")	Hooves opaque, horn pits appear, fine hair on eyebrows
150	3.4kg (6.5-10lbs)	30-45 (12-17.5")	Hair on eyebrows and lips, testes in scrotum, teats well formed
180	5-10kg (11-12lbs)	40-60 (15.5-24")	Hair on inside of ear, around horn pits, tip of tail and muzzle
210	8-18kg (17.5-40lbs)	60-75 (24-30")	Hair on metatarsal/carpal and phalangeal regions, on back, long hair on tip of tail
240	15-25kg (33-55lbs)	65-85 (26-34")	Fine short hair all over Incisor teeth not erupted
270	20-50kg (44-110lbs)	80-100 (32-40")	Hair coat complete and long Incisor teeth erupted

*From Roberts, 1971 and Hubbert et al., 1972

1 2

3 4

5 6

Fig 1 - Meconium staining of the haircoat of a fetus dying from intrapartum asphyxia.

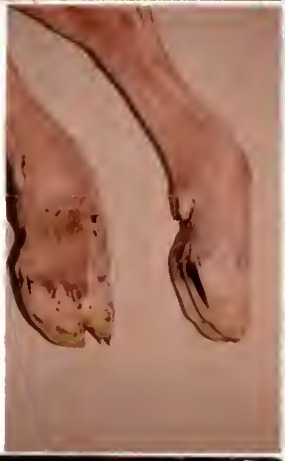
Fig 2 - Localized subcutaneous edema of head and submandibular space of a calf dying at the end of a birth of long duration. Note the swollen protruding tongue.

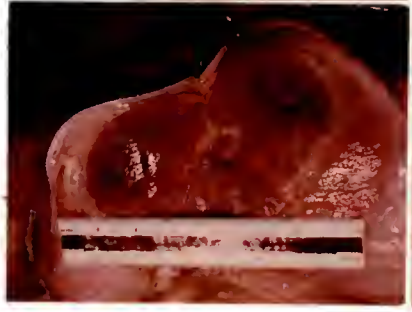
Fig 3 - Hooves of a calf that did not walk. Note the intact eponychium. Compare with Fig 4.

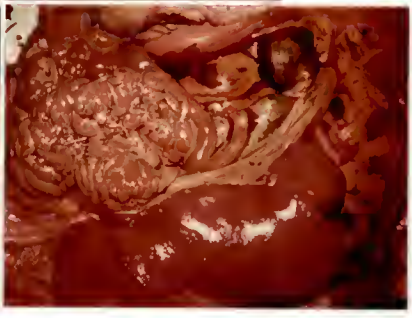
Fig 4 - Feet of a calf that stood and walked. Note the worn eponychium.

Fig 5 - Subcutaneous edema and hemorrhages in the rear muscles, partum death.

Fig 6 - Antepartum death -- note the generalized hemoglobin staining of the abdominal viscera in situ. The Hb imbibition imparts a uniform brownish-red to all viscera, muscles and fluids.







7	8
9	10
11	12

Fig 7 - Thoracic cavity of a partum death opened to reveal atelectasis of the lungs in situ.

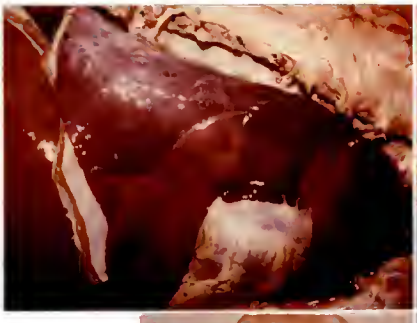
Fig 8 - Trachea of a calf dying from intrapartum asphyxia opened showing asperated meconium.

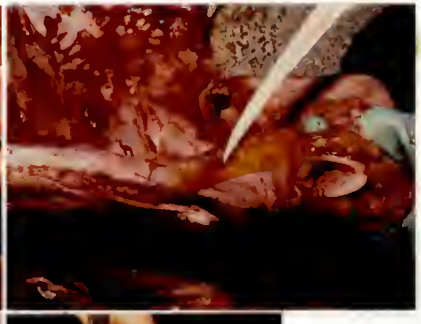
Fig 9 - Heart of a partum death dissected to show a patent ductus arteriosus.

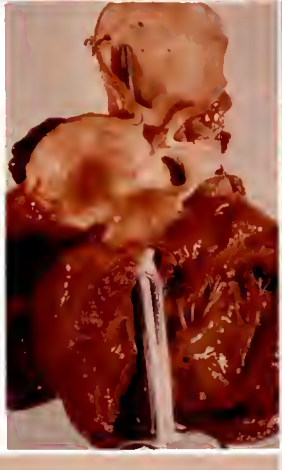
Fig 10 - Heart of a partum death dissected to reveal a patent foramen ovale.

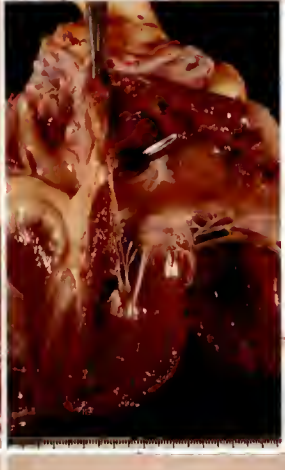
Fig 11 - Postpartum death - dissected umbilical cord to demonstrate a clot in an umbilical artery.

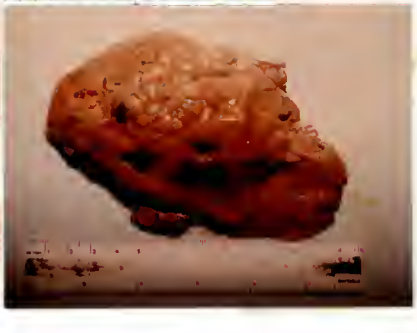
Fig 12 - Kidney of a newborn calf enclosed in fat - part of the neonate's energy reserves.











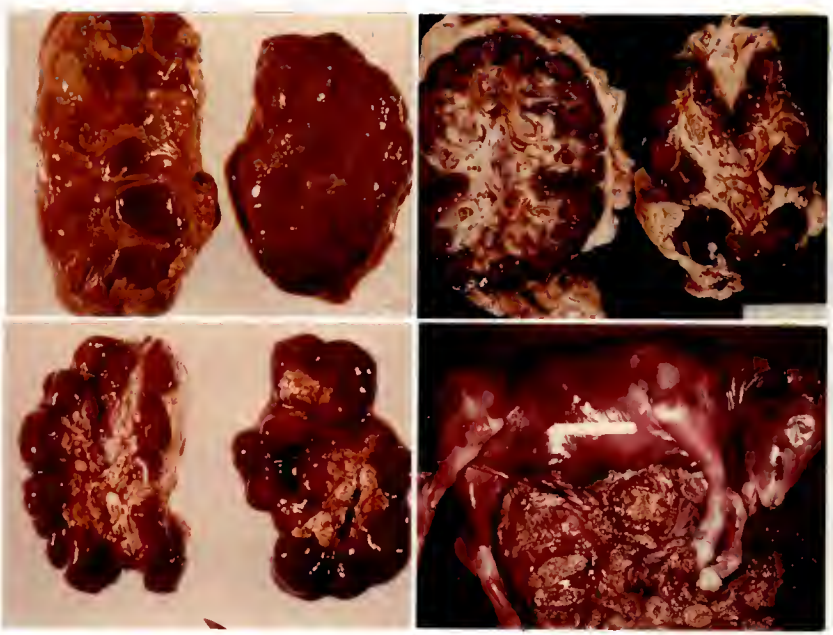
13	14
15	16

Fig 13 - Kidneys from two postpartum deaths. The kidney on the left illustrates moderate metabolism of fat compared to complete fat metabolism in the right kidney.

Fig 14 - Kidneys from a partum death without any autolysis of the renal cortex.

Fig 15 - Kidneys from two partum deaths -- the left shows moderate renal cortical autolysis compared to advanced autolysis in the right. Compare the kidneys to those in Fig 14.

Fig 16 - Antepartum death - aborted fetus with edema and generalized hemoglobin staining. Note the skin lesions and severe necrotic placentitis typical of mycotic abortion (Dr. C.A. Kirkbride, South Dakota State University).



VI. PERINATAL CALF MORTALITY IN KANSAS FLINT HILLS: GENERAL DISCUSSION

A systematic necropsy approach based on gross findings correlated with time-of-death (TOD) was used to examine and compare information between calves and ranches. TOD determination divided the calf deaths into antepartum, intrapartum, and postpartum. The study is one of few systematic studies where the entire spectrum of calf deaths within a population were examined. The study was not biased towards a specific cause such as abortion, dystocia, enteritis, pneumonia or congenital defects. The information gathered, although at time lacking in sufficient numbers, was representative of the perinatal calf mortality in the calf population in the Flint Hills of Kansas.

The systematic necropsy and TOD approach proved to be efficient for gathering, standardizing, and identifying the general and specific causes of PCM as well as the predisposing factors. By using standardized terms and definitions and five TOD classes, the approach and methods can be used by other researchers or cattle practitioners who wish to determine the incidence, distribution, and causes of PCM in specific regions or individual herds. By simply adding TOD determination to any perinatal necropsy and recording it, a practitioner will develop a better understanding of non-infectious factors and disease trends in perinatal calf problems in his client's herds. Progressive practitioners in the forefront of cow/calf health and production management can benefit greatly from this systematic approach. When all calves dying within a herd are necropsied not only

can assessment of the herd's reproduction efficiency be determined, but more importantly, perinatal problem areas can be specifically identified. Once definitely identified, more effective management and/or health programs can be designed and utilized. The old adage of "a problem accurately defined is half solved" is very applicable to herd health and production management.

Classifying dead calves into one of five functional TOD categories is relatively simple and easily determined. If it is impractical for a veterinarian to perform necropsy and TOD classification on a high percentage of dead calves then the ranch owners, managers, or responsible hired men could be quickly trained to make and record TOD determination. This will require them to be able to recognize localized edema, a clot in the umbilical arteries, hemoglobin staining, aerated or collapsed lungs, and degree of fat metabolism in a dead calf. Feedlot cowboys have long assisted and even performed necropsies on dead cattle when the consulting veterinarian was not available. Although they cannot be expected to do as complete or accurate an examination as a trained veterinarian, they can quickly learn to identify certain gross lesions and signs. This idea could also be applied to the cow/calf sector of beef cattle production. The veterinarian could then collect weekly reports and monitor perinatal calf problems on a ranch or a number of ranches in his practice area. From analysis of this basic information determination of the incidence and distribution

of PCM on a ranch may be made and updated. By comparing information from a ranch to its established goals or accepted "normals" in the area and using a statistical significance chart, problem areas could be identified early and corrected before the problem becomes a major economic loss. If problem areas would emerge, the consulting veterinarian would then have much of the information needed for rationale decisions to assist the rancher(s). A few complete necropsies performed by the veterinarian on selected calves may be necessary to confirm the diagnosis. If a definitive diagnosis cannot be made grossly by the veterinarian, the TOD classification is beneficial for determining what, if any, diagnostic tests may be necessary. By comparing the TOD of calves necropsied to a list of common causes of PCM within that TOD category, in the particular region, a list of probable causes may be made. From this list, an objective and cost efficient number of diagnostic tests could be performed.

Only in the last decade have techniques like these and others been used as part of herd health and production management programs. Now with the use of microcomputers, these programs can include a number of variables so that comparisons can be quickly made. The systematic necropsy and TOD approach lends itself easily to computerization as demonstrated in this study. It allows examination of the effects of many non-infectious and environmental influences on calf mortality and this is an area where our present

understanding is limited. PCM is a complex problem resulting from a variety of non-infectious and infectious causes and their interactions including management, climate, nutrition, behavior, infections, toxins, congenital defects, predation, genetics, and unknown factors. Without accurate definitions and a systematic approach, it is no wonder that the incidence of PCM has not improved appreciably over the past 50 years. This lack of improvement when compared to the advances made in husbandry, nutrition, preventive medicine, infectious disease, and therapeutic agents is disappointing.

Approximately two-thirds of the calf deaths in this study occurred during birth and the neonatal adaptive process. Non-infectious causes predominated with deaths from dystocia being the largest single cause of calf loss followed by neonatal weakness, the major cause of death during the first 24 hours of life. A high proportion of the calves dying of neonatal weakness had undetermined specific causes of death. Abortion was the major cause of antepartum deaths and even with a high level of diagnostic support many were undiagnosed. Many of the undetermined abortions may have resulted from non-infectious causes that are poorly understood or they may have been due to unknown factors. Complicated starvation from enteritis is the major infectious calf disease in beef cattle of this region. The interaction of poor colostrum intake and single or dual pathogen

invasion is important in the area of complicated starvation. Many factors may be involved with poor colostrum intake but one of the most obvious is lack of viability following dystocia. This is an excellent example of interaction of non-infectious and infectious factors resulting in perinatal calf mortality.

The number of viruses associated with neonatal calf infections continues to rise in direct proportion to the increased utilization of more refined diagnostic laboratory techniques. Increased utilization and understanding of fetal serology may prove to be the best way of identifying many of the infectious causes that presently are undetermined. ELISA testing shows great promise for practitioners as many of the tests can be performed quickly, accurately, and efficiently in the practitioner's laboratory and are economical.

Improved management, especially during the first week of life is essential for reducing calf losses and increasing production. Increased calf survivability may be increased by reducing environmental stress, a better understanding of parturition, better informed cow/calf management personnel, identifying breed differences, and utilizing planned cross-breeding programs. A 10% increase in total calf crop has a larger economic benefit to producers than does a 10% change in any other variable of a cow/calf operation. Increasing calf survivability should be the number one goal of every cow/calf operation. Increasing calf survivability is

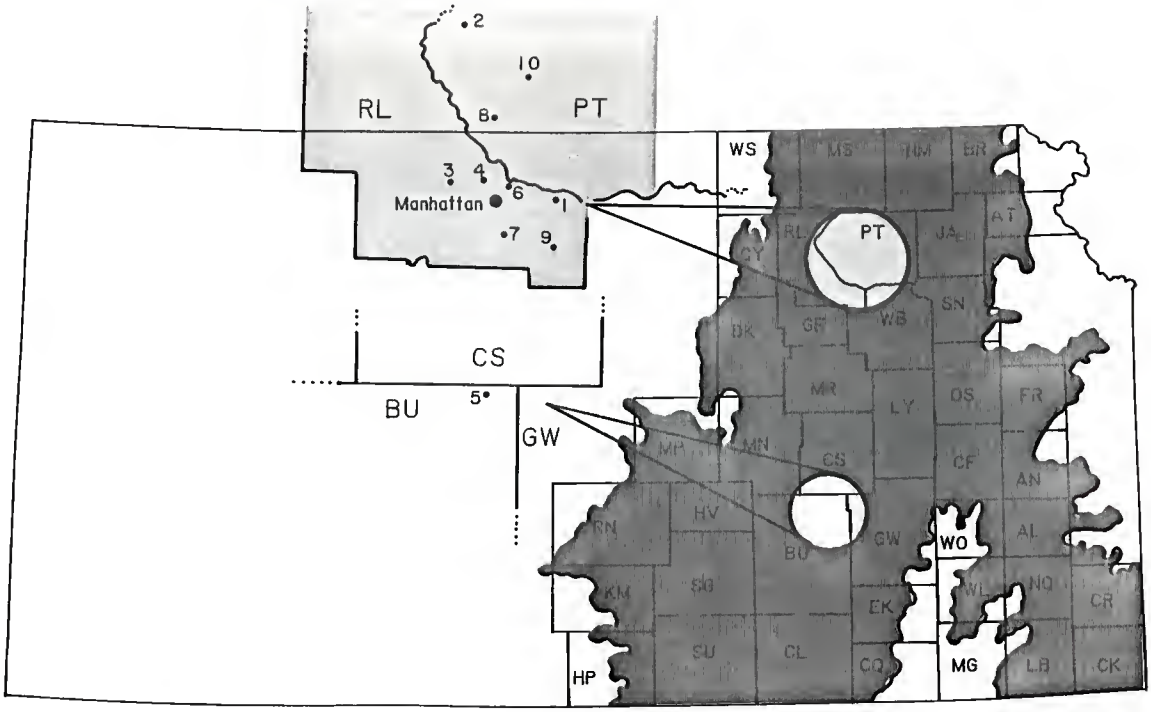
synonymous with improved production efficiency. Nationally if calf survivability could be increased a mere 1% this would result in an increase in income to U.S. beef cattle producers of at least 100 million dollars.

A perinatal calf mortality rate of 5% is considered to be economically acceptable. Rates of less than this are technically feasible but, unless cost-effective to producers, will not be achieved. Even in this area of the United States if the average PCM rate per herd could be lowered from 8 to 5%, this would result in an income increase of approximately $900 to 1,000 at weaning per 100 cows. Cow/calf production efficiency will not increase substantially until more work is done to define the specific problem constraints within geographic regions. Health and production management programs must be tailored to fit environmental, economic, and disease factors within any geographic regions. Cattle breeds and crossbreeding plans must also be determined with the intent of maximizing a given environment. However, regardless of location the key to continued improvement is in planning and prevention and not simply treating problems as they arise or are identified. Future perinatal calf investigations with close cooperation among various disciplines -- systems approach -- should help clarify the losses and causes of production efficiency. Specific areas in need of additional research identified during this study include:

1. Continuation of the present study.
2. Expansion and coordination of the project with other geographic areas for comparison.
3. Specific environmental factors and their interaction as they relate to and influence PCM.
4. Development of total herd production management programs centered around maximum calf survival.
5. Predisposing factors and causes of neonatal weakness.
6. Causes and influences of natal nonviability.
7. Causes of non-infectious abortion.
8. Better understanding of the mechanism and process of parturition in beef cattle.
9. Physical and hormonal interactions affecting neonatal viability and colostrum intake and antibody absorption.
10. Perinatal manifestations of experimental Haemophilus somnus infection.
11. Further determination of the importance of pathogen specific fetal antibodies.
12. Multiple pathogen ELISA testing plates for abortion diagnosis.
13. Multiple pathogen ELISA testing plates for diagnosing single and dual enteric infections.
14. Research on breed and breeding strategies based on maximizing various environments and increasing calf survivability.
15. Developing a PCM statistical significance chart for identifying problem areas in a herd early.

VII. APPENDICES

APPENDIX 1: Map of Kansas Illustrating the Flint Hills Region and the Counties Participating in the PCM Project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APPENDIX 2: Perinatal Calf Mortality Herd Survey Form

PERINATAL CALF MORTALITY HERD SURVEY
Department of Pathology - Dr. Stanley Dennis
Department of Surgery & Medicine - Dr. Mark Spire
Department of Animal Sciences & Industry - Dr. Billy Able
Manhattan, KS 66506

1. Name _____
2. Mailing Address _____
3. Approximately what percent of your beef cowherd calves in the fall? _____ In the spring? _____
4. From your spring calving herd, when was your first calf born in 1982? _____ Approximately what percentage of your spring calves were born within 30 days of this calf? _____
   When was your last spring calf born in 1982? _____
5. Do you individually identify calves at birth? _____
6. How do you decide which cows will be culled from your herd?
   Open _____
   Last calf _____
   Poor condition _____
   Poor weaning weights _____
   Bad eyes _____
   Bad udders _____
   Diseases _____
   Structural defects _____
   Disposition _____
   Age _____
7. If available, actual weaning weights of 1982 calf crop.
   Bulls _____ Steers _____ Heifers _____
8. About how long was your 1982 breeding season (dates)?
   _____
9. How many heifers _____, cows _____, and total females _____ were bred last year?
10. How many calves did you wean last fall? _____
11. Have you noted any abortions since breeding last year? _____ How many? _____

12. Did you notice any bull problems last year? Yes __ No __
    If yes, what kind of problem? _____
    _____
    Was breeding delayed from normal? Yes _____ No _____
13. Were there any unusual circumstances, such as extreme weather, health problems, etc, that you were aware of in 1982? _____
    _____
    _____

14. What vaccinations did you use last year on your:
    Cows _____
    Replacement heifers _____
    Calves _____
15. Did you pregnancy check last year? _____ If so, how many heifers _____, cows _____, total herd _____ were pregnant?
16. In your wintering program:
    Do you feed high protein cakes? _____
    Do you feed high energy cakes? _____
    Do you feed grain? _____
17. What <u>principal</u> roughages did you use last winter?
    Rough grazing _____ Corn stalks _____
    Sorghum stalks _____ Grass hay _____
    Alfalfa hay _____ Corn silage _____
    Sorghum silage _____ Cane hay _____
    Others (please specify) _____
18. What mineral and salt supplements do you feed during the:
    Summer? _____
    Winter? _____
    How are they fed? _____
    Included in other feeds _____ Blocks _____
    Loose free-choice _____
19. Do you provide extra feed in winter for:
    Open heifers? _____
    Young cows? _____
    Old cows? _____

20. Do you start supplementing winter feed in:
    Oct _____ Nov _____ Dec _____ Jan _____
    Feb _____ As needed _____
21. Do you purchase replacement females?
    None _____ Open heifers _____ Bred heifers _____
    Open cows _____ Bred cows _____
22. At what age do you first calve heifers? _____
    (month of age)
23. What percent of your heifer crop did you save in 1982 for replacements? _____
24. On what basis do you select replacement heifers?
    (If more than one goal, rank 1st, 2nd, etc.)
    Performance of her sire _____
    Performance of her dam _____
    Breeder reputation _____
    Type or conformation _____
    Weaning weight _____
    Yearling weight _____
    Weight for age _____
    Frame _____
    Conception _____
25. How many females left your herd in 1982?

Number	Approx Date	Reason	Cow or Heifer	Open or Pregnant
_____	_____	_____	_____	_____
_____	_____	_____	_____	_____
_____	_____	_____	_____	_____

26. Where did you obtain your present bulls?
    Raised own _____ Breeder _____
    Salebarn _____ Swap with neighbor _____
    Test Station _____ Lease _____
    Other (please specify) _____
27. What do you consider when selecting a bull?
    (Rank 1, 2, 3, etc.)
    Breeder reputation _____ Type or conformation _____
    Performance of her sire _____ Performance of her dam ____
    Birth weight _____ Weaning weight _____
    Yearling weight _____ Weight for age _____
    Frame size _____ Cost _____

28. Do you semen check your bulls prior to breeding season? _____ Were any found to be infertile? _____
29. What age bull do you prefer to choose when buying? _____
    How many years do you generally keep your bulls for breeding purposes? _____
30. Do you weigh calves at birth? Yes ____ No ____ if yes, please complete.
    What was the average birth weight for heifer calves? ____
    What was the average birth weight for bull calves? _____
31. How many heifers calved? _____
    How many cows calved? _____
32. How many of your heifers needed assistance while calving? _____ How many of your cows? _____
    How many calves were lost during or within 5 days of birth? _____
    How many calves were lost during or within 28 days of birth? _____
33. Did you treat for internal parasites in 1982? _____
    If yes, what did you use?
    Injectable _____ Paste _____ Drench _____ Feed _____
34. What was your fly control program in 1982?
    None ____ Spray _____ Back rubbers _____ Ear tags ____
    Other (please specify) _____
35. What was your grub and lice control program in 1982? Give approximate dates of application.
    Cows _____
    Bulls _____
    Calves _____
36. How many calves were lost at or within 1 month of weaning in 1982? _____
    Causes?

APPENDIX 3: Necropsy Request Form

DEPARTMENT OF PATHOLOGY
KANSAS STATE UNIVERSITY

Clinic Case No. _____

Necropsy No. _____

REQUEST FOR NECROPSY EXAMINATION (To be completed and **signed** only by the clinician)

History attached: Yes / No

Species _____ Breed _____ Sex _____ Age _____ Wt. _____

Owner _____ Address _____

Death: Date _____ Time _____ A.M. P.M. Died ☐, Euthanatized ☐, Method _____

Antibiotic/Chemotherapeutic Treatment Yes ☐, No ☐, Unknown ☐ _____

Requests by clinician _____

Clinical Diagnosis _____

_____

Permission for Necropsy Granted _____ Date _____
Signature of Clinician

DIAGNOSTIC LAB REQUEST: Bacteriology ☐, FA ☐, Other ☐ _____ Lab. No. _____

Blood ☐ _____	Intestine ☐ _____	Liver ☐ _____
Spleen ☐ _____	Lung ☐ _____	Stomach ☐ _____
Kidney ☐ _____	Heart ☐ _____	_____ ☐ _____

Organism Suspected/Comment: _____

_____

GROSS PATHOLOGICAL FINDINGS

Condition: good, fair, poor, _____; Post-mortem decomposition: nil, little, moderate, advanced, _____

SIGNIFICANT GROSS FINDINGS: _____

_____

_____

_____

_____

_____

_____

_____

_____

TENTATIVE GROSS DIAGNOSIS: _____

Students _____ Pathologist _____

PROCEDURES PERFORMED: Histopath. ☐, Parasitology ☐, Chemistry ☐, Photography ☐

1. Diagnostic Lab (White)
2. Pathology (Canary)
3. Clinic (Pink)

APPENDIX 4: Perinatal Calf Necropsy Form

KANSAS STATE UNIVERSITY
PERINATAL CALF NECROPSY EXAMINATION FORM

Necropsy Case No. _____ Clinic Case No. _____
Owner I.D. _____ Ear Tag No. _____ Oate _____
Breed _____ Sex _____ Age _____ Wt. _____ Actual/Estimated
Comments: _____

EXAMINATION OF THE CALF

___ Condition: good/fair/poor/emphysematous/ dehydrated; < 5%/5-10/>10%
___ Carcass Oamage: antemortem/postmortem
___ Hooves: walked/did not walk
___ S/C edema: generalized/localized; < 1cm/1-2cm/>2cm; clear/blood tinged; fluid/gelatinous; submandible/cervical/gluteal/extremities/forelimb/hind limb

___ Muscles: atrophic/Hb stained/hemor./ congested/pale
___ Tongue: swollen/ulcerated
___ Oral cavity and Gingiva: hyperemic/ hyperplastic/ulcerated
___ Thyroid: enlarged/small
___ Lungs: atelectic/edematous/hemor./ pneumonic/autolytic/pluritis
___ % inflated 0/10/20/40/60/80/90/100
___ Thymus: small/hemor.
___ Heart: defect/↑Rt/↑Lft/ epi. hemor./ endo. hemor.
___ Ouctus: closed/open; < 2mm/2-4/4-6/>6mm
___ Hydrothorax: clear/blood tinged/clot/ fibrin; <50ml/50-500/>500ml
___ Ribs cage: fractures/hemor./adhesions
___ Spine: defect/abscess/fracture/luxation
___ Ascites: blood tinged/clot/fibrin; <100ml/100-1000/>1000
___ Umbilical Artery: thrombus/empty/abscess
___ Limbs & Joints: Enlarged/defect/abscess/edema

___ PMA: nil/little/mod./adv.
___ Malformation: single/multiple
___ Umbilical ends: squared/pointed; wet/dried

___ Scrotum: abscess/hematoma/edema
___ Bladder: empty/full and distended
___ Renal Autolysis: nil/little/mod./marked
___ Adrenals: enlarged/small
___ Fat Metabolism: nil/partial/generalized
___ Liver: congested/contracted/autolytic/ nodular/rupture/necrotic foci
___ Abomasum: hemor./erosions/ulcers
___ contents = empty/milk/grass/hair/mud/ mucus/water/medication
___ S. Intest.: hyperemic (Sl,Mod,Mrk)/ hemor./autolytic/dilated/defect
___ contents = normal/fluidy/pasty/fibrin/ bloody/medication
___ L. Intest.: hyperemic (Sl,Mod,Mrk)/ hemor./autolytic/dilated/defect
___ contents = meconium/normal/fluidy/ pasty/fibrin/bloody/medication
___ Mes. L.N.: enlarged/edematous/hemor.
___ Brain: defect/congested/hemor./edematous/ meningitis
___ Spleen: contracted/flabby/enlarged

EXAMINATION OF THE PLACENTA

___ Not presented
___ PMA: nil/little/mod./adv.
___ Cotyledons: enlarged/edematous/necrotic/reddish/yellowy/mottled
___ ICA: thickened/edematous/hyperemic/hemor./adventitious/necrosis

---

SPECIAL PROCEOURES

___ Immunoglobulin concentration
___ Oarkfield
___ Photographed
___ FA/ELISA for: _____
___ Serology for: _____
___ Smears: _____
___ Histopath tissues: _____

___ Bacteriology
___ Virus Isolation
___ Toxicology

SUMMARY

Type of Oeath: Oevelopmental/Infect./ Non-Infect./Unknown
T.O.O. Classification: _____
Tentative/Final Oiagnosis: _____

APPENDIX 5: Criteria for Determining the Time-of-Death Classification of Calves Necropsied

Time of Death Classification Relative to Birth*

1. Antepartum Deaths
   APD 1...dead for a long time
   APD 2...dead for > 12 hours
   APD 3...dead for < 12 hours

2. Partum Deaths
   Early in a birth of:
   PD 1...moderate duration
   PD 2...long duration
   Middle in a birth of:
   PD 3...moderate duration
   PD 4...long duration
   End of a birth of:
   PD 5...moderate duration
   PD 6...long duration
   PD 7...short duration

3. Postpartum Deaths
   Immediate PP deaths (IPPD)
   PPD 1...did not breathe
   PPD 2...breathed, did not walk — body fat not metabolized
   PPD 3...walked, did not feed — body fat not metabolized
   PPD 4...food not beyond small intestine — body fat not metabolized
   Delayed PP deaths (DPPD)
   PPD 5...breathed, did not walk — body fat partially metabolized
   PPD 6...walked, did not feed — body fat partially metabolized
   PPD 7...food not beyond small intestine — body fat partially metabolized
   Late PP deaths (LPPD)
   PPD 8...walked, did not feed — body fat fully metabolized
   PPD 9...food not beyond small intestine — body fat fully metabolized
   PPD 10..body fat not metabolized — food has passed through whole of G.I. tract
   PPD 11..body fat partially metabolized — food has passed through whole of G.I. tract
   PPD 12..body fat fully metabolized — food has passed through whole of G.I. tract

*From McFarlane (1965), modified by Dennis (1981)

Antepartum Death (APD) Classification*

APD 1	Premature gestatinnal age and mummification (Dead for a long time)
APD 2	Premature gestational age and generalized hemoglobin staining (Dead for more than 12 hours)
APD 3	Premature gestational age and yellow edematous fluid or no edema (Dead fnr less than 12 hours)

Partum Death (PD) Classification*

Class	Time of death	Duration of birth	Umbilical artery clot	Degree of local edema	Degree of renal autolysis
PD1	early	mnderate	absent	little nr none	moderate
PD2	early	long	ahsent	little or nnne	marked
PD3	middle	moderate	absent	mnderate	moderate
PD4	middle	long	absent	marked	marked
PD5	end	moderate	absent	moderate	none
PD6	end	long	absent	marked	nnne
PD7	early	short,	absent	little or none	none

Postpartum Death (PPD) Classification*

Class	Umbilical artery clot	Breathed	Walked	Nursed	Food passed through GI tract	Body fat metabolized
PPD 1	+	-	-	-	-	none
PPD 2	+	+	-	-	-	none
PPD 3	+	+	+	-	-	none
PPD 4	+	+	+	+	-	none
PPD 5	+	+	-	-	-	moderate
PPD 6	+	+	+	-	-	moderate
PPD 7	+	+	+	+	-	moderate
PPD 8	+	+	+	-	-	complete
PPD 9	+	+	+	+	-	complete
PPD 10	+	+	+	+	+	none
PPD 11	+	+	+	+	+	moderate
PPD 12	+	+	+	+	+	complete

+ = signs present

- = signs not present

*Dennis (1984)

APPENDIX 6: Protocol for Tissue and Specimen Collection

Perinatal Calf Mortality

PROTOCOL FOR:

Necropsy Tissue and Specimen Collection

I. Materials

- Culture Swabs 2
- Whorl Pak's 3 or 4
- 6cc Syringe 1
- Red Top Blood Tube I
- Container of 10% NBF 1
- Cardboard Sample Holder 1

II. Procedure

1. Blood Collection: RED TOP TUBE full of blood then take to Path. room K217.
   - With the calf in left lateral recumbency, reflect the right forelimb.
   - Use the 6cc syringe without the needle to collect the blood from the axillary blood vessels(May need to put pressure on thorax to get blood out).
   - Remove rubber stopper from tube and transfer blood.
   - Label tube with necropsy case number.
2. Bacteriology Swabs: Swab of THORAX and ABDOMEN for Diagnostic Lab.
   - Label two culture swab covers with necropsy case number, date and location of specimen taken (THORAX or ABDOMEN).
   - Take swab of thorax and abdomen when first opened.
   - If any lesions are noticed swab them instead.
3. Bacteriology Tissues: ABOMASUM or SMALL INTESTINE depending on TOD class.
   - If calf is given TOD classification of APD, PD or PPD 1-4; ligate both ends of the ABDMASUM and place into a whorl pak.
   - If calf is given TOD classification of PPD 5-I2; ligate both ends of a piece of SMALL INTESTINE (or amy intestine that appears inflamed) and place into a whorl pak.
   - Label whorl pak with necropsy case number, date, tissue taken (abomasum or small intestine) and the word "Bacteriology".
4. Fluorescent Antibody Tissues: LIVER, LUNG, SPLEEN on all, SMALL INTESTINE on some.
   - LIVER, LUNG & SPLEEN sections will be taken from all calves for tests for IBR, BVD, PI-3 and Blue Tongue viruses.
   - Calves given TOD classification of PPD 5-I2 will also have a piece of SMALL INTESTINE included for Rota and Corona Virus tests.
   - Put tissues in whorl pak and label with necropsy case number, tissues contained, date and tests required.
5. Virus Isolation Tissues: LIVER, LUNG, SPLEEN, KIDNEY on all, SMALL INTESTINE and CONTENTS on some.
   - LIVER, LUNG, SPLEEN and KIDNEY sections will be taken from all calves.
   - Calves given TOD classification of PPD 5-I2 will also have a piece of SMALL INTESTINE and CONTENTS included or put in a separate whorl pak.
   - Label whorl pak with necropsy case number, date, tissues contained and the words "Virus Isolation".
6. Histopathology Tissues: LIVER, LUNG and THYMUS on all calves.
   - Thin sections of LIVER, LUNG and THYMUS will be placed in the container of ID% NBF and labeled with the necropsy case number, date, owner and the name "Cain".
7. Dther Tissues or Tests: As directed by lesions or the Pathologist incharge.

APPENDIX 7: Calf Master File Data Entry Sheet

Kansas State University
Perinatal Calf Mortality Project

CALF MASTER FILE DATA ENTRY SHEET

1. Year_____ (82 or 83)

2. Calf Necropsy Number_____ (DDD1 to 9999)

3. Ranch I.D. Number: 01 02 03 04 05 06 07 08 09 10 11 12 13 14 15

4. Breed: H(Hereford) A(Angus) S(Simmental) X(Crossbred) F(Holstein) O(Other)

5. Sex: B(Bull) S(Steer) H(Heifer)

6. Weight:_____ (DD1 to 999)

7. Dam's Age: O(Unknown) 1(2 yr. or less) 2(3 yrs old) 3(Greater than 3 yr.)

8. Ig Concentration:_____ (DD.01 to 50.00) or (Ranges: eg. 5-15)

9. T.D.D.:_____ (APD-1 to 3, PD-1 to 7, or PPD-01 to 12)

10. Cause: D(Developmental) I(Infectious) N(Non-infectious) U(Unknown)

11. FA/EIA: IBR= 1+, 1- ; BVD= 2+, 2- ; PI-3= 3+, 3- ; BT= 4+, 4- ; Corona Virus= 5+, 5-
Rabies= 6+, 6- ; DN599= 7+, 7- ; Rota Virus= 8+, 8- ; E. coli K88= 9+, 9-

12. Bacteriology:_____ (1-26 see Diagnostic lab inputs)

13. Serology:_____ (see Diagnostic lab inputs)

14. Virus Isolation: 1(BVD) 2(IBR) 3(PI-3) 4(BT) 5(Chlymidia) -(Negative)

15. Darkfield: Campylobacter like= 1+, 1- ; Leptospira like= 2+, 2- ;
Trichomonia like= 3+, 3-

16. Diagnosis:_____
(Limited to 50 spaces)

APPENDIX 8: Samples of Reports sent to Contract Calf Herds

KSU
KANSAS STATE UNIVERSITY

Department of Pathology

College of Veterinary Medicine
Veterinary Medical Center
Manhattan, Kansas 66506
913-532-5634

January 21, 1983

Dr. Miles McKee
Purebred Beef Unit
Dept. of Animal Science and Industry
Kansas State University
Weber Hall
Manhattan, KS 66506

RE: Perinatal Calf Mortality

Dear Dr. McKee:

The following is a summary of the necropsies performed on the three calves that you submitted to Kansas State University's College of Veterinary Medicine for the year 1982.

1. Calf #82-2gg: A one day old 85 lb. female Simmental calf was presented on February 12, 1982. This calf was taken by cesarean section and manually fed colostrum but exhibited signs of weakness and death. Postmortem examination revealed signs of fetal stress, neonatal weakness and nonviability. Microscopic examination of the lungs showed large areas of incomplete inflation (congenital atelectasis). Fluid and meconium were also seen within the lung alveoli. No evidence of toxic or infectious agents were found.
   Diagnosis: Neonatal weakness due to aspiration pneumonia and improper inflation of lungs (congenital atelectasis).

2. Calf #82-313: A full term 58 lb. newborn male Simmental calf was presented on February 14, 1982. Postmortem examination revealed signs of fetal distress during birth and neonatal weakness after birth. Brain hemorrhages were observed grossly. No evidence of toxic or infectious agents were found.
   Diagnosis: Death shortly after birth due to neonatal weakness from birth trauma.

3. Calf #82-44g: A three day old 120 lb. male Simmental calf (#15-P) was presented on March 4, 1982 with a history of central nervous system (CNS) problems and death. Postmortem examination revealed icterus, peritonitis, abnormal and excess cerebrospinal fluid, and intracranial hemorrhages and congestion. Microscopic examination of the lungs revealed a mild interstitial pneumonia with areas of fluid and meconium

inhalation. Also seen were areas of incomplete lung inflation (congenital atelectasis). Microscopic examination of the brain showed a severe meningoencephalitis with bacterin present. Bacteriologic examination revealed the bacterium to be of the type known as E. coli.
Diagnosis: Acute meningoencephalitis and septicemia due to a bacterium called E. coli.

We were unable to arrive at the time of death and probable cause of death in all three of the calves submitted for necropsy.

During the past year your herd was part of about 1,700 cows, from various ranches in this area, participating in a study of Perinatal Calf Mortality. The information gained from these herds helps to assess, not only the causes and possible preventive means of calf mortality in your herd, but also in determining relative causes of calf mortality in this region. With this information we hope to evaluate individual herd calf mortality rates and to pin-point problems or diseases for the purpose of protecting present and future calf crops.

As of now, we have added 1,050 more cows to increase the resource pool for the 1983 calving season. This is still only a fraction of the total number of cows in your area. In order to obtain records that are truely representative of a herd's or the area's perinatal calf mortality, postmortem examination of every dead calf (including stillbirths) is a must. If you know of any rancher or farmer that would be willing to participate in this program this year please have them contact Dr. Mark Spire, Assistant Professor of Surgery and Medicine, College of Veterinary Medicine, Veterinary Medical Center, Manhattan, Kansas, 66506, or call (913) 532-6187.

Your cooperation in this program is appreciated and we hope that this information will be of value to you.

Sincerely,

S. M. Dennis, Head, Department of Pathology

Don Cain, DVM/MS Student

KSU
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913-532-5634

February 2, 1984

Purebred Beef Unit
c/o Dr. Miles McKee
Dept. of Animal Science & Industry
Weber Hall
Kansas State University
Manhattan, KS 66506

RE: 1983 Perinatal Calf Mortality Summary

Dear Miles:

Thank you for your participation in this project this past year. The information gained from your herd along with the other ten herds in the area was very helpful in allowing us to access the incidence and distribution of calf mortality, as well as specific causes. These and other large scale studies patterned after this one are essential in giving us accurate information and tools needed to help cow/calf producers identify problem areas and come up with economical solutions.

The following is a summary of diagnoses reached on the calves that you submitted to Kansas State University, and also included is a comparison of the percent distribution of calf mortalities in your herd to those of the percent distribution of calf mortalities of the combined ten herds in this area.

- Total of 22 calves presented.

- Summary of Diagnosis:

Calf ID#	Diagnosis
83-0383	Abortion suspected due to Chlamydia infection
83-0445	Abortion of unknown cause
83-0554	Dystocia (difficult birth) and birth trauma
83-0634	Dystocia (difficult birth) and anoxia (suffocation)
83-0648	Dystocia (difficult birth) and anoxia (suffocation)
83-0836	Dystocia (difficult birth) due to fetal weakness
83-1385	Birth trauma: Ruptured liver
83-0430	Birth trauma: Spinal column luxation
83-0771	Aspiration pneumonia from drowning
83-0430	Neonatal weakness from fetal anemia
83-0444	Neonatal weakness from shock
83-0647	Neonatal weakness from cold exposure

Calf ID#	Diagnosis (continued)
83-0771	Neonatal weakness from respiratory failure
83-0599	Neonatal weakness of unknown cause
83-0967	Acute death with cerebral and pulmonary edema
83-0854	Complicated starvation due to colibacillosis
83-1048	Complicated starvation due to colibacillosis
83-1049	Complicated starvation due to colibacillosis
83-1100	Complicated starvation due to E. coli septicemia
83-0837	Complicated starvation due to E. coli and Cl. perfringens enteritis
83-0812	Complicated starvation due to Rotavirus enteritis
83-0813	Starvation

- Distribution of calf mortality:

Death	Your Herd Distribution on 22 calves	Area Distribution on 89 calves
Before birth (APD)	9.1%	5.6%
Early during birth (EPD)	4.6%	3.4%
Late during birth (LPD)	13.6%	29.2%
Immediately after birth (IPPD)	31.8%	22.5%
Late after birth (LPPD)	40.9%	39.3%

To complete our studies and to make the above data more accurate so you can use it as a management tool, we need two more pieces of information from you. Please answer the two questions at the bottom of this page, detach, and send them to us in the self-addressed, pre-stamped envelope enclosed.

Due to your cooperation and support of this project this past year, we have chosen your herd to remain as one of the area project herds for another year. Therefore, we will provide free diagnostic services to you on all calf mortalities you have this spring. We are pleased that you understand the importance of having every dead calf you have brought to KSU for evaluation.

If you have any questions about the information in this letter (or in submitting calves this spring), please contact Dr. Mark Spire at the College of Veterinary Medicine at 532-6187 or Dr. Bill Able in the Department of Animal Science at 532-6131.

Sincerely,

Dr. A. C. Strafuss
Project Head, Department of Pathology

Don Cain
DVM/MS Student

(Please complete and return)

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1. Number of calves that died this past spring and were not sent to KSU: _____

2. Total number of cows and heifers calved out this past spring: _____

KSU
KANSAS STATE UNIVERSITY

Department of Surgery and Medicine

College of Veterinary Medicine
Veterinary Medical Center
Manhattan, Kansas 66506
913-532-5690

May 10, 1984

Galen Fink
Purebred Beef Unit
Weber Hall
CAMPUS

Dear Galen:

The following is a breakdown of the calves I have identified as being from the Purebred Beef Unit and submitted for the perinatal calf mortality project.

24 Feb 84 - male - abortion - BVD infection

12 Mar 84 - bull - heifer 89K - possible rotavirus, negative pathogenic E. coli

30 Mar 84 - female - died during birth - bled through navel

10 Apr 84 - cow 10N - calving problem - prolonged delivery

16 Apr 84 - female - calving problem - liver injury

16 Apr 84 - female - acute pneumonia - may be IBR

24 Apr 84 - male - calving injury - broken back

29 Apr 84 - male (1 mo) - blackleg

Unknown date (late Feb 84) - female - Angus - birth trauma

Unknown date (12 Mar 84) - male - Angus (10 days) - scours and starvation (calf quit nursing) - unknown cause

Unknown date (mid March 84) - male - SM - inhaled fluids at birth - pneumonia

Unknown date (mid March 84) - female - SM - exposure death

Sincerely,

Mark F. Spire, DVM, MS

MFS/pb

APPENDIX 9: Five Functional Time-of-Death Categories

Gross criteria of the five functional categories of time-of-death summarized from McFarlane's classification of 21 classes (McFarlane, 1965).

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1. Antepartum Deaths (APD 1-3)

   Characterized by fetal death before abortion or parturition. Gross findings include absence of clots in the umbilical arteries, clear to blood-tinged edema of the subcutis and serous cavities, generalized hemoglobin (Hb) staining, renal cortical autolysis, atelectatic lungs, mummification, and incomplete development.

2. Early Partum Deaths (PD 1,2,3 and 7)

   Characterized by intrapartum fetal death with little or no signs of viability. Major gross findings include lack of thrombi in umbilical arteries, no localized or generalized subcutaneous edema, and no Hb staining. Other findings may be moderate to marked renal cortical autolysis, meconium standing of the haircoat, partial lung aeration, and petechiae on pleura, peri- and epicardium, and thymus.

3. Late Partum Deaths (PD 4-6)

   Characterized by fetal death but with positive signs of viability. Major gross findings include no thrombus in

umbilical arteries, and presence of localized subcutaneous edema of the head, limbs or perineum. Other findings often seen are similar to those found in early parturient deaths.

4. Early Postpartum Deaths (PPD 1-7)

Characterized by signs of surviving birth with a functional heart, unsuccessful neonatal adaptation, and death within 48 hours of birth. Major gross findings include soft to firm thrombi in the umbilical arteries, and little to no body fat metabolism. Other findings may include partial, complete or no lung aeration; may or may not have walked; meconium may be present or absent, may or may not have fed; and little or no intestinal absorption of milk.

5. Late Postpartum Deaths (PPD 8-12)

Characterized by signs of being liveborn, usually successful neonatal adaptation but failure to sustain life resulting in a delayed death (> 48 hours to 28 days). Major gross findings include a firm thrombus in the umbilical arteries, and moderate to complete body fat metabolism. Other findings often include all of those associated with early postpartum deaths except that the calves had usually nursed, and no meconium as food has passed through the digestive tract.

PERINATAL CALF MORTALITY IN THE KANSAS FLINT HILLS

by

Donald Verne Cain Jr.
B.S., University of Nebraska, 1981
D.V.M., Kansas State University, 1984

ABSTRACT OF A THESIS

submitted in partial fulfillment of the

requirements for the degree

MASTER OF SCIENCE

Department of Pathology

KANSAS STATE UNIVERSITY

Manhattan, Kansas

1985

Production efficiency is the most important factor in determining profitability of a commercial beef cow-calf enterprise and the goal is to obtain a live marketable calf from every cow bred each year. A major constraint in realizing this goal is perinatal calf mortality (PCM). It is unlikely that PCM losses will be reduced until the problem is clarified and all factors defined.

This study was undertaken to investigate PCM in the Flint Hill area of Kansas over a three-year period (1982-1984). The main objective was to determine, define and clarify the incidence, distribution, and general causes of PCM in beef herds by systematic necropsy and time-of-death (TOD) classification.

Two populations of calf deaths were examined and compared, 155 contract and 82 non-contract, total of 237 calves necropsied. Calf deaths from 3,934 females expected to calve in contract herds were examined resulting in 7.99% PCM. Incidence was significantly influenced by year ($P \leq .06$) with years 1982 and 1983 being significantly different ($P < .05$). Cow herd size and type of operation also influenced the PCM incidence. The incidence of PCM for the type of operation with respect to cow herd size was significantly different ($P < .05$), with a highly significant negative correlation between incidence of PCM and cow herd size ($P < .001$). There were highly significant differences between calf populations for distribution by time-of-death

($P \leq .0001$) and general causes of PCM ($P \leq .0001$). The contract calf population distribution of PCM was antepartum 6.5%, early partum 7.7%, late partum 28.4%, early postpartum 27.1%, and late postpartum 30.3%. Distribution was significantly influenced by year ($P < .01$) and cow herd size ($P < .0005$). Specific categories of TOD had highly significant differences when compared to general causes of PCM ($P \leq .0001$). General causes of PCM were not influenced by year or type of operation but approached significance with cow herd size ($P \leq .09$). Dystocia was the most frequent condition associated with PCM (25.8%), neonatal weakness was second with a relatively high frequency of 21.9%, and disease complicated starvation was third at 16.8%. Noninfectious conditions were the most prevalent causes of PCM (59.4%) with 55.4% occurring during the parturient period. Infectious causes accounted for 24.5% of all losses with the majority (78.9%) occurring during the late postpartum period. Natal nonviability and neonatal weakness were identified as important conditions associated with PCM with a high frequency of undetermined causes.

Incidence of infectious and undetermined causes of PCM was 24.5% and 16.1%, respectively. Increased use of laboratory tests decreased the incidence of undetermined diagnoses and also increased the percent of viral causes of PCM diagnosed. Bacterial culturing was the most efficient diagnostic tool used followed by single sample postmortem

serology of the calf, darkfield examination, immunofluoresence and ELISA testing, and virus isolation. Serologic results were significantly related to TOD ($P \leq .0001$). Leptospirosis and infectious bovine rhinotracheitis were the major causes of abortion; Escherichia coli and rotavirus were the major causes of enteritis; E. coli was the major cause of septicemia; and Clostridium perfringens was the major cause of acute infectious deaths. Bovine virus diarrhea and Haemophilus somnus were the most common causes of infectious neonatal weakness and were also associated with a wide range of neonatal infectious conditions. Dual infections were found in 2.5% of calf deaths and 8% of infectious conditions.

Non-infectious causes were determined in 108 calves (45.6%); most prevalent during the early partum, late partum, and early postpartum periods with 55% of all non-infectious PCM occurring during parturition. Non-infectious incidence of PCM was influenced by year (environmental factors) and size of cow herd (management factors) ($P \leq .09$). Crossbred calves were less associated with non-infectious perinatal mortality than purebred calves (15.2% vs 84.8%). Male calves were associated with a higher incidence ($P \leq .01$) of PCM during the early and late parturient categories compared with females (85.7% and 65.3%, respectively, for males vs. 14.3% and 34.7%, respectively, for females). There was no significant difference between weights of the two sexes. Mean calf weight was 77 ± 30 lbs. with a wide

range (5 to 275 lbs.). Dystocia and severe birth trauma in combination accounted for 51.9% of all of the non-infectious causes. Most prevalent non-infectious causes were birth trauma 43.5%, anoxia/respiratory failure 15.7%, and exposure 10.2%. Congenital defects were observed in 5.2% of the contract calves examined; visceral defects 46%, skeletal 23%, central nervous system 15%, and multiple defects 15%. Various cardiac defects accounted for one half of the visceral defects, and internal hydrocephalus was the only CNS defect.

It was concluded that the approach of systematic necropsy and TOD was efficient for identifying the general and specific causes of PCM as well as the predisposing factors. Future perinatal calf investigations will require, in addition, a farming systems approach with close cooperation between various disciplines for increasing production efficiency. A number of areas requiring additional research were identified.